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Southwestern Medicine

OFFICIAL ORGAN OF
ARIZONA STATE MEDICAL ASSOCIATION
NEW MEXICO MEDICAL SOCIETY
EL PASO COUNTY (TEXAS) MEDICAL SOCIETY
THE MEDICAL AND SURGICAL ASSOCIATION
OF THE SOUTHWEST

Volume XV JANUARY, 1931 No. 1

ANNUAL SUBSCRIPTION $2 SINGLE COPIES 25 CENTS

Entered at the Postoffice at Phoenix, Arizona, as second class matter.
"Acceptance for mailing at special rate of postage provided for in section 1103, Act of October 3, 1917, authorized March 1, 1921."

THE MANAGEMENT OF LOBAR PNEUMONIA

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(Read before the Annual Meeting of the Medical and Surgical Association of the Southwest, held in El Paso, Texas, Nov. 6-8, 1930.)

These observations are based on a series of 602 consecutive cases treated in the pneumonia ward of the Los Angeles General Hospital during the past four and one-half years. The service was shared by Dr. D. D. Comstock, professor of medicine of the College of Medical Evangelists. We were ably assisted by four junior attending physicians of the hospital staff. This series does not include cases of lobar pneumonia in children, as these are cared for on the pediatric service of the hospital.

The etiology, symptomatology, and diagnosis will not be discussed except to state that in all cases of this series the diagnosis was confirmed by x-ray films taken on the ward by a portable apparatus, or by autopsy. The word "management" in my title is used in preference to "treatment" because it is my belief that lobar pneumonia is a self-limited disease, the outcome of which, except possibly in a relatively small percentage of cases of types 1 and 2, cannot be altered by any specific treatment known at the present time. I do feel, however, that in a certain number of cases the outcome can be favorably influenced by careful nursing and symptomatic treament, which conserve the strength of the patient.

If the mortality of this series seems high to those of you who treat lobar pneumonia in private practice, please remember that these patients come largely from the very lowest stratum of society. Hoboes and Mexicans of the poorer class comprised a considerable portion of the group. There were many chronic alcoholics and others whose resistance to infection had been lowered by fatigue, exposure and lack of food. Often the patients were moribund on admission to the hospital and died within eighteen hours; yet our mortality rate for the entire group is 24.7 per cent. which I believe compares favorably with the figures from other large charity hospitals. In private practice a much smaller group of patients cared for according to the plan which I shall outline, showed a mortality rate of less than 10 per cent.

I shall discuss the management of lobar pneumonia under the following general headings: (1) General hygienic measures, (2) the treatment of symptoms, (3) specific therapy, and (4) the treatment of complications.

Under hygienic measures it should be needless to mention the desirability of a light, cool, well-ventilated room; an open porch is no longer considered necessary or even desirable for the pneumonia patient. The diet should consist of liquids and such soft foods as can be taken with the least effort on the part of the patient. The calories can be augmented by adding lactose to the fruit juices given. Fluids should be pushed to the patient's capacity in order to aid in the elimination of toxins.

Rest is of the greatest importance. All too often the patient is disturbed by visitors, too frequent chest examinations by the physician, or the ministrations of an overzealous nurse. Particularly tiring is the daily cathartic or enema, which the pneumonia patient, whether at home or in the hospital, seldom escapes. For the past year no patient on my service has been given either cathartic or enema during the critical stage of his illness. When this order was first given, our head nurse, a veteran of many battles with the pneumococcus, threw

up her hands in horror, but now, without a qualm, she allows the patients to go four or five days, or even a week, without an evacuation. When a patient gets well enough to worry about this apparent neglect he is given a mild cathartic or an enema. Since the adoption of this plan, I believe we see fewer patients with abdominal distention, probably the most dreaded symptom to occur in pneumonia.

Pain and cough often interfere with rest. These two symptoms are usually the only ones to require attention in the early days of the attack. Chest pain is best relieved by fixation. A binder pinned tightly about the thorax during expiration usually answers the purpose better than adhesive plaster strapping. Morphine may be needed for a day or two, seldom longer.

Cough can best be controlled by codeine in adequate doses, preferably administered hy hypodermic injection. The patient's stomach should be spared the various cough mixtures often prescribed.

Cyanosis may be an early and alarming symptom. It denotes anoxemia and should be relieved when possible. This can usually be accomplished by the administration of oxygen. The ideal method is by the oxygen chamber, which, however, is not usually available because of the expense entailed. The earlier cases of our series were given oxygen by means of a nasal catheter, strapped in position; more recently a small tent devised by Dr. A. E. Guedel has been used. This tent fits over the head and shoulders of the patient and is provided with a glass window. By a special valve attached to a large tank of commercial oxygen, the gas can be administered almost constantly at a cost of about seven dollars for twenty-four hours. Occasionally patients complain that the air in the tent becomes uncomfortably warm. This objection can be eliminated in most cases by increasing the oxygen flow.

Meteorism, as previously stated, is a symptom of the gravest import. It probably means splanchnic paralysis resulting from toxemia and may be compared to the alarming paralytic ileus which occasionally follows abdominal operations. In this condition the abdomen becomes drum-like; the diaphragm is pushed upward, thus embarrassing further the already labored respiration. The only therapeutic measure which in our experience has seemed to be of value in this condition, is the free administration of surgical pituitrin in doses of ½ to 1 c.c.

Vasmotor paralysis is probably the usueal cause of death in lobar pneumonia. The signs are falling blood pressure, moist cool skin, livid color and rapid, irregular pulse. Except for the dyspnea usually present, the appearance of the patient is much like that of one in profound surgical shock. It is the custom in many hospitals to use digitalis early in the course of the disease in the hope of preventing this circulatory failure. In an effort to determine the value of this procedure, our ward was divided into two services, to which cases were admitted alternately. On one service all cases of lobar pneumonia were rapidly digitalized as soon as the diagnosis was made. On the other service no patient recevied digitalis unless there were indications of previous myocardial failure, or auricular fibrillation occurred. In the first series, digitalization was accomplished by giving to a patient of average weight 6 grains of the powdered leaf every six hours for three doses, then 1½ grains twice a day until discontinued. In only two cases was there evidence of poisoning by the drug. In all, 239 cases received digitalis; of these, 81, or 33.4 per cent, died. On the other service there were 363 cases, none of which were digitalized; of these, 68, or 18.7 per cent, died. Recently Niles and Wyckoff[4] have published the results of a more complete study of the same nature on 825 cases observed at Bellevue Hospital. In this series the mortality rate was 7.7 per cent higher in the digitalized group. These results have convinced us that digitalis should not be administered to pneumonia patients except in those cases in which some definite indication, such as auricular fibrillation or preexisting myocardial failure, is known to be present. Furthermore, since the publication of Niles' report we have concluded that we are not justified in continuing this investigation.

The drugs upon which we rely for stimulation are caffein and ephedrin. Glucose intravenously, and even blood transfusion, may be of value in some cases.

Since 1897, when Washburn first immunized a horse and used the serum in the treatment of pneumonia in man, there have been numerous reports in the medical literature on the efficacy of specific treatment. To be of benefit, the serum must be given early and in large amounts. Only those cases caused by the pneumococcus types 1 and 2, are benefited. These comprise about fifty per cent of all cases. Cecil[2], Cole[3], Park[4] and Finland[5] have each reported a large series of adequately controlled serum-treated cases. The largest series, reported by Cecil from the Bellevue Hospital, comprised 885 cases. The mortality in the untreated cases was 39.2 per cent; in the serum-treated cases, 30 per cent, or more than 5 per cent higher than in our 602 cases treated without

serum. About two years ago we administered Felton's concentrated serum for types 1 and 2, to a group of seventeen patients. This group was "hand-picked," so to speak, being young or middle-aged adults who on admission had not been ill more than forty-eight hours. After the usual precautions the serum was given at once and continued only if typing proved the case to be suitable. Of the first twelve cases treated, ten recovered and we became quite enthusiastic. However, four of the next five cases died, giving a mortality for the series which compared unfavorably with our general average. The cost of Felton's serum is so great that the hospital authorities dicouraged its further trial. Following this experience, Mulford's polyvalent vaccine was tried for a time with no better success. Although as yet not entirely convinced of its value, I would use serum in an early case proved by typing to belong to group 1 or 2, provided there were no history of asthma or other allergic discase to a surgical service. When fluid in sensitization.

Of the serious complications of lobar pneumonia, empyema is, of course, the most frequent. We try to make the diagnosis early, but we are in no hurry to transfer the case to a surbgical service. When fluid in the pleural cavity is suspected, an x-ray film is ordered. If this confirms our suspicions, the aspirating needle is used to determine the character of the fluid and to withdraw a sufficient amount to relieve pressure symptoms, if these are present. Nothing more is done until we are quite sure that the acute pneumonic process in the lung has subsided. Then, and only then, is the case turned over to the surgeon for closed drainage or rib resection. Many a life has been lost by hasty surgical interference in empyema. Occasionally, repeated aspiration of the pus results in cure. Antiseptics such as genian violet have proved of doubtful value in the infected pleural cavity.

Abscess of the lung was formerly thought to occur not infrequently following pneumonia. It is probable, however, that in the majority of instances these cases were lung abscess from the start and due to some agent other than the pneumococcus. I do not recall a case of abscess in which blood culture showed the pneumococcus at any stage of the illness. Regardless of its etiology, acute abscess of the lung should be treated conservatively at first. Anything more radical then bronchoscopic drainage should be avoided until the case "cools of." Probably half of these cases recover without surgery.

Pneumococcus meningitis is a comparatively rare complication. We have treated two cases by repeated spinal puncture, drainage, and injection of ethylhydrocupreine; both died. Recoveries have been reported following spinal drainage with the injection of serum.

The idea prevails, at least among the laity, that pneumonia is less frequent and less severe in communities like Southern California, which enjoy a climate comparatively free from sudden changes and severe cold. It may be true that the incidence is somewhat less in this climate, but I am convinced that mild climate does not mean a mild disease. Pneumonia is apparently as severe in our Southwest as in New England.

To summarize: We believe that lobar pneumonia is a self-limited disease, the duration of which cannot be modified by any means known at present, with the possible exception of a small percentage of cases (types 1 and 2) which may be benefited by the early and frequent use of specific serum. In our experience digitalis has been worse than useless. Codeine, caffein, ephedrin and pituitrin are the only drugs which we have found to be of apparent value. Oxygen relieves cyanosis and restlessness; it should be used early and freely. The routine use of enemas and cathartics is to be condemned. Attention to ventilation, diet and rest are measures which may turn the tide in favor of our patient, and the most important of these is rest.

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Sodium 2-Oxo-5-Iodo-Pyridine-N-Acetate (Introduced as Uroselectan).—The Council on Pharmacy and Chemistry publishes a preliminary report on sodium-5-iodo-2-pyridon-N-acetate) sodium 2-oxo-5-iodo-pyridine-N-acetate (also designated as a new substance for use in the radiographic visualization of the urinary tract which was introduced under the name Uroselectan. The Council reports that the product is a definite chemical substance which promises to be an advance in the radiographic visualization of the urinary tract, though its indications, advantages and limitations are at the present time not fully established. The Council considers the product suitable for experimental use by those who are versed in the technique of urologic examinations. It publishes a concise and carefully considered statement of the introduction of the drug and the evidence for its use, prepared for the Council by Drs. Braasch and Bumpus. (Jour. A.M.A., November 8, 1930, p. 1425.)

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THE DIAGNOSTIC CRITERIA OF HEART DISEASE

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(Read before the Annual Meeting of the Medical and Surgical Association of the Southwest, at El Paso, November 7, 1930.)

Failure is the gloomy specter that haunts the future life scenes of the individual with heart disease. Whatever the heart lesion, providing it is organic, the ultimate outcome, though often postponed for years by careful therapy, is usually incapacitating pain or swelling. Death comes either suddenly with a breast pang or less insidiously with increasing dyspnea or orthopnea, congestion, cyanosis and edema. The exact mechanisms by which these states are brought about are not yet clearly established. This field of study offers much for investigation. Certain clinical facts are known and guide us in our attempts to forestall the vicious circle of so-called decompensation. Intercurrent infection, undue strain, frequently repeated insults, psychic emotional upsets, mechanical embarrassments, adverse physical and climatic conditions all help to precipitate the break in compensation. There is no recognizable change in the pathological anatomy suddenly present in the heart, but the precipitous disturbances in the cardio-respiratory functions are often extreme. The interesting facts and theories concerning these mechanisms warrant more detailed discussion which cannot be gone into at this time. The diagnosis of heart disease in the presence of failure is relatively simple, it is the recognition early in the incipiency that is essential.

The ultimate fate of the patient with organic heart disease as popularly conceived is sufficient to strike fear into any one afflicted. The diagnosis of heart disease in the minds of many is the equivalent of a death sentence. This fate seems inevitable, inexorable, swift, precipitous, immediate, and hideous to some. The verdict should therefore be reserved and not pronounced until there is not a shadow of a doubt, only after searching study, seasoned deliberation, and even then never abruptly or uncermoniously, but with tact and discretion after careful consideration of the patient's psychological reactions. In fact, it is the part of wisdom to avoid the discussion of the findings and to refrain from the diagnosis of cardiac abnormalities in any but those of impassive indifference. Carelessly used, loosely descriptive expressions, as "valve leakage," "heart damage," "dropsy" and "angina pectoris," thoughtlessly meted out are most harmful. The temperamental, worrisome type of individual is apt to conjure up for himself a terrible imaginary or phantom symptom complex and the phychic insults of such a brain storm of the self-sentenced individual are more detrimental and demoralizing than are the actual concurrent physical sufferings.

To avoid such distressing incidences and unnecessary anguish it is not only desirable but necessary to establish some more or less dogmatic criteria. The physician must have a clear conception of what constitutes heart disease and what signs he is going to require as evidence thereof. Certain physical abnormalities, such as serious mechanism disturbances, significant thrills and murmurs, changed and adventitious heart sounds, enlargement, hypertension and arteriosclerosis are of definite diagnostic and prognostic significance.

The usual abnormal signs we must admit are only evidences of conduction, valvular, aortic and vascular lesions, but from the type and extent of such damage we can fairly accurately deduce the degree and the type of pathology present in the heart muscle and also often by inferences determine the etiological agent. This is especially true when the cardiac affection has been, as it so often is, the result of syphilitic, rheumatic, or streptococcic infection. These diseases are notorious for the fact that when they have produced cardiac valvular or aortic lesions they have also concurrently damaged the heart muscle. This myocardial damage may remain latent for many years but eventually symptoms and signs referable to it will present themselves in the inevitably developing clinical picture.

The added burden of the defective valvular or aortic mechanism must be recognized as a contributing factor to the ultimate fate of the case. Physical strains, especially if repeated, are poorly borne, add insult to injury and serve to hasten the onset of failure with its vicious circle of distressing symptoms and signs. Infections, anemia, chemical and physical changes in the blood and the vascular changes of advancing years are much more poorly tolerated and the cardiac cripple must be guarded against them and the advance of the same as much as it is possible to do so. This is one of the chief functions of the attending physician who is managing the case.

The present-day physician must, or should, have a clear conception of what constitutes heart disease, the daignostic signs of the condition the rational scientific in-

terpretation of the various findings, and the prognostic significance of these physical abnormalities. We can with a reasonable degree of accuracy determine and differentiate definitely the grave from the insignificant lesions; we know the possible complications, the usual progress of the various lesions, and the limitations to be placed upon the patient with a symptomatic organic heart disease. The conditions are rationally and dogmatically defined, even though the direct specific evidences of myocardial damage, before the advent of symptoms of insufficiency, are rather obscure and the functional tests still relatively crude and inaccurate. The exact histological picture that the heart muscle would present is indeed quite indeterminable.

Nevertheless, it should no longer be that "Truly fortunate are they who keep aloof from the stethoscope of the auscultator," as Austin Flint wrote in 1862. His extensive and profitable experiences in the Charity Hospital at New Orleans and his teachings had not yet been broadcast and as completely and widely appreciated as they are at present. The intervening years with the contributions of Mackenzie, Lewis, Wenckebach, Christian and many others, have given us information such that after the study of a case we can, with careful phychologic approach, usually accurately and reassuringly inform a patient of his condition without unduly alarming him. We can manage his case and direct his mode of living so as to add years of productivity and comfort and often even carry him through the average span of years of human lifetime.

The simple bedside methods, in the hands of the average informed physician, will yield the necessary critical clinical information upon which an accurate diagnosis and prognosis can be made, in the great majority of cases. The common laboratory procedures must be resorted to for confirmatory evidence. The history and the physical examination, but especially the latter, may be relied upon for the differentiae and omens of functional or organic heart disease cases.

THE CLINICAL HISTORY

The story that the patient with heart disease offers may, unfortunately often, be related verbatim by one with a cardiac neurosis, neurocirculatory asthenia or the effort syndrome. This subjective evidence must therefore always be of secondary or corroborative diagnostic value. Symptoms appear only when the heart muscle begins to lag in the performing of its job. That is, only when there is a failure or myocardial insufficiency of some degree. The characteristic complaints of pain, shortness of breath and swelling of the legs can be produced by other conditions, but, in the presence of heart disease, they indicate the extent of cardiac reserve. Pain in the substernal area and down the arm or under the right costal border, from the stretching of the Glissonian capsule of the engorged liver, dyspnea or orthopner, cyanosis or edema, are the symptoms and signs of ischemia, fatigue or exhaustion of the heart muscle.

The present illness in a reliable history is, in general, a fairly good index of the functional state of the circulation. The complaint of pain, especially when graphically described as the classical radiating breast pang of Heberden, is most significant, especially in one of stolid temperament in the middle decades of life. Under such circumstances the typical story is usually sufficient for a diagnosis of heart disease with anginal failure, even in the absence of physical findings. This, however, is the only exception to the rules to be elaborated herein.

The past history often reveals important information as to the original etiological factors and the symptom-precipitating circumstances. Information of this sort is useful in the formation of a prognosis and in the subsequent management of a case. A denial of venereal infection, however, must not deter one from making a diagnosis of syphilis of the aorta in a middle-aged man with a free aortic regurgitation or the signs of an aortitis and dilatation, in the presence, or even the absence, of other clinical or laborator data, when the less common other causes are eliminated. Likewise, a negative history of the rheumatic infection, as acute multiple arthritits, chorea, acute tonsillitis or growing pains, does not discount the diagnosis of rheumatic heart disease in the presence of a definite mitral stenosis. Exopthalmic goiter and other diseases of the thyroid, or other serious infections or chronic diseases, are of significance as contributory affections. The limits of this are defined by the history as to whether the patient is able to carry on his usual activities (Class I), or is unable to carry on his ordinary physical activity (Class II), in which he is either slightly limited (a) or greatly limited (b) or is unable to do anything whatsoever and must remain in bed or in a chair (Class III). (American Heart Association Classification of Myocardial Insufficiency). All this information, valuable as it is, remains subjective, and practically no unequivocal data can be derived from this

source. For reliable criteria for the true indications of heart disease, we must, therefore, turn to the other source, namely, the objective findings.

THE PHYSICAL EXAMINATION

Certain physical signs constitute the reliable diagnostic criteria of organic heart disease with all its grave potentialities of failure.

These physical signs, if they are to be considered absolute signs of heart disease, must be such that each single one in itself indicates the presence of actual or potential myocardial changes that sooner or later lead to congestive or the anginal type of heart failure. These signs must also be such and so inclusive that the absence of each and every one of them is proof that the patient is not suffering from heart disease. Any such index of signs must of necessity be rather dogmatic and may at times lead to slight error. On the whole, however, it is more satisfactory to be somewhat dogmatic than to be constantly vacillating and unsure of one's position. The diagnostic criteria of heart disease, that have been more or less generally accepted in cardiological circles, will be presented and discussed.

I. SERIOUS CARDIAC MECHANISM DISTURBANCES THAT INDICATE HEART DISEASE.

Auricular fibrillation, flutter, heart block and alternation, when definitely or permanently established, are produced only in damaged hearts. These significant disturbances of cardiac action may be elicited by an examination of the radial or carotid pulse and the apex beat with vagus, exercise or amylnitrite. The complete arrhythmia persisting and increasing as the rate rises to over 120 or 140; the rapid regular rhythm sharply but temporarily influenced by vagus pressure; the slowed regular rhythm increased by a multiple or uninfluenced by exercise; and the regularly recurring weak beats unaccompanied by pauses, are usually sufficient eveidence for a diagnosis of the disorders. The persistent presence of any one of the disturbances is most significant in that each is always preceded by, initiated by and accompanied by serious myocardial damage. Clinical or bedside means are usually sufficient to establish fairly accurately the diagnosis, with the possible exception of an occasional instance of atypical flutter and heart block. Electrocardiographic tracings will determine the exact nature of rhythm disturbances. Blood pressure studies or tracings, noting the alternate higher and lower levels of the systolic arterial blood pressure, establish definitely a record and confirm the suspicion of an ominous pulsus alternans, which is the sign of a gravely weakened myocardium.

II. SIGNS OF VARIOUS CHRONIC CARDIAC VALVULAR LESIONS THAT EVIDENCE HEART DISEASE.

Diastolic, mitral and aortic thrills and murmurs. Thrills are the palpable counterpart of loud murmurs. The latter are gravely significant only when diastolic in time, except for the occasional rare congenital systolic ones. A slow diastolic vibration is felt in the presence of an audible, rumbling, apical diastolic murmur, the characteristic physical findnigs in a patient with a well-developed mitral stenosis. Mitral stenosis is a valve lesion which spells previous rheumatic valvulitis and, consequently, rheumatic heart disease, with or without the history of previous infection. In rheumatic fever the myocardium is always directly affected to a greater or less extent. Focal and disseminated scars found in the auricular and ventricular muscle are proofs of the widespread involvement of these structures. With the added embarrassment of mechanical defects in the mitral or aortic valve, the heart is bound to give way eventually under the added strain.

The high-pitched basal diastolic murmur transmitted from the second right to the third left intercostal space and on toward the apex or axilla, so characteristic of a very free aortic regurgitation, signifies old rheumatic heart dsiease in the young individual, or syphilitic heart disease if the patient is middle-aged, or arteriosclerotic disease in senility. In vascular syphilis the root of the aorta is usually affected and the process in the wall frequently encroaches upon the orifices of the coronary arteries. Narrowing of these cuts down the blood supply to the heart, which is further and probably primarily impaired by the low diastolic blood pressure. Thus the coronary blood flow is reduced to a very low ebb. Besides this grave nutritional disturbance, the unusual infiltrated and fibroid lesions complete the evidence of direct and indirect involvement of the myocardium by the syphilitic virus. Syphilitic vascular disease without heart disease is inconceivable.

III. ABNORMAL AREAS OF AORTIC AND CARDIAC, DULLNESS; AORTITIS, ANEURYSM, HYPERTROPHY OR DILATATION OF THE HEART.

Enlargements of the great vessels, or of the heart itself, invariably interfere with the essential blood supply of the myocardium, and the encroachment sooner or later leads to insufficiency and congestive failure. The signs of aortitis, the increased retro-

manubrial dullness, must be accompanied by a pathologic ringing, metallic or tambourlike aortic second sound in the presence of a normal blood pressure. Further dilatation to the point of production of aneurysm gives rise to increasing dullness, abnormal pulsations, diastolic shocks, or rebounds, tracheal tugging, unequal brachial blood pressures and radial pulses and transmitted heart sounds. Besides these, we find an extensive vascular damage, and all taken together constitute the manifestations of vascular syphilis, especially when accompanied by sharply accentuated reverberating, liquid aortic second sounds in the presence of a normal blood pressure. Syphilitic aortitis, aneurysm, especially the latter, do not produce as profound changes in the heart as are present in aortic regurgitation. Nevertheless, the changes are significant. Traumatic, arterio-venous aneurysm, unrepaired for many years, results in myocardial damage in the hypertrophied heart. Fluoroscopic examination and teleoroentgenograms, especially with lateral and left and right oblique views, are generally employed to corroborate the clinical diagnosis.

Clinically recognizable increase in the size of the heart means a pathological heart muscle which sooner or later will fail, principally because its blood supply cannot keep pace.

The increased area of cardiac dullness is likewise not sufficient evidence, in itself, of cardiac enlargement. It may be produced by a pericardial effusion, but under such circumstances it is of about equal significance.

Visible displacement to the left of the midclavicular line of the cardiac apex is generally conceived to be the most definite, almost infallible clinical or bedside sign of cardiac enlargement, in the absence of dullness in the opposite chest. For the most part, the circumscribed apex impulse is the best criterion of the size of the heart on the left border of dullness, which sometimes is difficult to make out. This, for the most part, corresponds to the left border of the normal or moderately enlarged heart, and in very large hearts does not quite indicate the full extent of the left border of the heart to the left. If this extension is more than 15 cm. beyond the midsternal line, if definitely outside the midclavicular line, if there is no gross evidence of any massive collapse of the left, or air or fluid in the right, pleural cavity, cardiac enlargement is present. Dilatation and hypertrophy of the heart are incompatible with continued normal function and integrity of the myocardium, because, sooner or later, with aging if from no other reason, the circulation of the heart itself is embarrassed. There can be added, to substantiate the signs, the graphic evidence of fluoroscopic findings, or the diagrams or teleoroentgenograms, which yield absolutely conclusive evidence of an exact measurement of the shadow of the enlarged heart. The evidences of etiological factors producing the cardiac enlargement may be present. These usually are either the valvular lesions, the sclerosis and tortuosity of all peripheral vessels, increased blood pressure, evidences of renal damage. Hyperthyroidism, or hyperthyroidism, may be revealed, further substantiating the diagnosis of heart disease. Chronic adhesive pericarditis usually escapes detection.

The increased area of cardiac dullness, abnormal areas of dullness in the regions of the great vessels, are usually corroborative of previously elicited signs, such as visible and abnormal pulsations of the cardiac impulse, substantiating the diagnosis of aneurysm or myocardial disease. Pericardial adhesions anchoring the heart to the chest walls produce physical embarrassment to cardiac action, increasing as time goes on and, at the same time, constricting, and by pressure decreasing, the circulation and predisposing to circulatory embarrassment, damage, and eventually failure.

IV. CHRONIC, TOXIC, VASCULAR, METABOLIC AND RENAL DISTURBANCES INVARIABLY DAMAGE THE HEART.

Chronic hypertension, chronic nephritis and chronic hyperthyroidism are recognized etiological factors in the production of advanced heart disease, and therefore their presence alone over a period of time would be taken as indirect evidence of myocardial damage. One cannot be absolutely certain just how this damage is brought about, but evidently there is quite a strain in the burden of continued high blood pressure. There is a characteristic narrowing of the arterioles all over the body, producing nutritional changes in the heart muscle as well as in other muscle elsewhere, with ultimately the concomitant arterial changes. The circulation of retained metabolic by-products, incomplete and abnormal endocrine, especially thyroid secretions, inevitably produces direct chemical changes on the heart muscle cells. Shifts in the acid-base, electrolyte, blood protein, albumin and globulin levels, and osmotic changes, are still theoretical consideration and explanaton only. The extra burden of increased circulatory efficiency, against greater or less odds, results in heart muscle changes recognized as pathologic cardiac hypertrophy.

Cardiac damage may not always be see-

ondary but may be initiated by the same factor or infection which produces the recognized hypertension, nephritis or hyperthyroidism, as presenting symptoms.

A chronic hypertension, or hyperpiesia, is one which persistently remains 30 or more millimeters of mercury above the accepted average normal for the patient's age and sex.* In the female a temporary postmenopausal increase in blood pressure is not so ominous. Some accept anything over 160 millimeters of mercury systolic and 90 millimeters diastolic as hypertension; others put the critical level as 180 systolic and 100 diastolic.

V. GENERALIZED VASCULAR CHANGES OR LOCALIZED CORONARY THROMBOSIS MEANS HEART DISEASE.

Widespread arteriosclerosis demonstrable only peripherally, and the vascular enlargement and elongation effecting an increased tortuosity of the vessels, may be considered as evidence of a generalized process. The aortic root and, consequently, the orifices of the coronary arteries, are practically never spared in widespread arteriosclerosis. On the other hand it is common to have these central arteries involved with relatively little definite change in the peripheral ones. The inevitable nutritional disturbances impair the structure, as well as the function, of the myocardium. The changed coronaries can no longer supply the threefold volume demanded on exertion and, consequently, there results an ischemia cardis. There may often be, with the cardiothoracic distress, spasms or coronary obstructions, but no unequivocal signs evident on ordinary physical examination.

Heart disease is always present in the presence of such vascular diseases. These may be lacking and subjective evidence alone, an excruciating substernal pain radiating into the arm, accompanied by blood pressure changes and vasomotor disturbances, must occasionally be accepted.

The story of one acute attack of severe persisting precordial pain—followed by dyspnea, frothy blood-tinged pulmonary-edema fluid, cyanosis, distant heart sounds, a friction rub, leukocytosis and fever—indicates that an infarction of the myocardium has taken place as the result of localized, often minimal, coronary changes. The coronary thrombosis usually so damages the heart that anginal or congestive failure appears sooner or later.

COMMENTS

It is, of course, sufficient for the diagnosis of heart disease to find one of these reliable signs as an index. However, the rule is that not only the one presenting in a given case need be depended upon. Usually there are several corroborative findings. When a patient, however, does not present any one of these ten physical indices, no matter what the symptomatology may be, one can feel quite secure in stating that the patient is not suffering from heart disease. In the absence of each and every one of the cardinal signs or predisposing state, the individual cardiac symptoms, regardless of their intensity, must be considered as a part of functional rather than organic heart disease. The prognosis accordingly would be much better, and any claim for disability could not be upheld. Rare electrocardiographic abnormalities, evidence of prolonged auriculoventricular conduction time, high-grade intraventricular defective conduction, may in themselves be indicative of grave myocardial damage, but usually these cases present one, and often more, of the clinical bedside findings. Clinical findings and conclusions are substantiated and corroborated by this graphic means.

In summary, then, the conception of what is meant by the term "heart disease" and the basis for the diagnosis of an organic circulatory disturbance is highly desirable from the patient's standpoint as well as from the physician's.

A diagnosis of "heart disease" should be made only in those who present the reliable criteria or physical signs which can be taken to indicate that the heart muscle itself has suffered definite structural impairment such that its reserve is diminished. The damaging effects of infection, allergy, anoxemia, exhaustion, strain, under nutrition, or developmental disturbances. Mechanical embarrassments may actively contribute to the eventual picture but are always of secondary moment. The damaged heart muscle is such that the signs and symptoms of myocardial insufficiency, incompetency, namely, congestive or anginal heart failure, will appear in the presence of such precipitating factors as undue exertion, intercurrent toxemia, or the inevitable vascular and circulatory changes and complications of advancing hypertrophy and age and the concomitant embarrassment of a coronary circulation.

The signs which indicate this are arrang-

*The generally accepted normal systolic blood pressure level is calculated on the basis of 120 at 20 years with 1 millimeter rise for every two years above this age, thus putting the normal level at 135 millimeters of mercury at 50 years. The significance of decidedly subnormal levels in advancing years has not been absolutely established.

ed in groups as follows: First, Auricular fibrillation, flutter, pulsus alternans and heart block. Second, Diastolic, mitral and aortic thrills and murmurs. Third, Increased dullness of aortic dilatation, aneurysm and cardiac enlargement. Fourth, toxic, hyperthyroidism, metabolic or renal disturbances, chronic hypertension and nephritis. Fifth, Generalized arteriosclerosis.

SODIUM AMYTAL FOR THE PREANESTHETIC PREPARATION OF SURGICAL CASES

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(Read before the Annual Meeting of the Medical and Surgical Association of the Southwest, held in El Paso, Texas, Nov. 6-8, 1930).

The derivatives of barbituric acid have been used for many years as hypnotics and sometimes as anesthetics. In order to clarify the relationship between the descriptive clincal term and the synonymous trade name of the barbituric acid derivatives which have assumed some significance in medicine, Lundy[1] has prepared the following list.

Iso-Amylethyl Barbituric Acid—Amytal.
Isopropylethyl Barbituric Acid—Ipral.
Phenylethyl Barbituric Acid—Luminal.
Cyclohexenylethyl Barbituric Acid—Phanodorn.
Ethylbutyl Barbituric Acid—Neonal.
Phenylmethyl Barbituric Acid—Rutonal.
Diethyl Barbituric Acid—Veronal or Barbital or Malonal.
Allylisopropyl Barbituric Acid—Allonal or Numal.
Diallyl Barbituric Acid—Dial.
Isobutylallyl Barbituric Acid—Sandoptal.
Diethyl Barbituric Acid and
Allylisopropyl Barbituric Acid—Veronal and Allonal, called Somnifene.
B-Brompropenylisopropyl Barbituric Acid—Noctal.
B-Brompropenylisobutyl Barbituric Acid—Pernocton

The sodium salts of these derivatives of barbituric acid are preferable to the acid itself for many reasons, chiefly because of the solubility.

Sodium iso-amyl-ethyl barbituric acid (sodium amytal) is furnished by the Eli Lilly Company in ampules containing 16 grains. This is dissolved in 10 c.c. of sterile distilled water making a 10 per cent solution. This solution shows a hydrogen ion concentration of between 9.5 and 9.8, which gives a maximum anesthetic effect with a minimum degree of toxicity. This solution can be given intravenously, orally, subcutaneously or intraspinally. Our experience with sodium amytal has been entirely with the intravenous route. The drug has been used only as a preanesthetic hypnotic and not as an anesthetic.

This report is in no way an attempt to compare sodium amytal with other barbituric acid derivatives but only to record our experience with a method which we consider a great advance in the preanesthetic preparation of surgical patients. There has been no fatality in this series traceable to the sodium amytal.

This report comprises 200 cases. One hundred and twenty-five cases were private patients and seventy-five cases were from our clinic at the Los Angeles General Hospital. The sodium amytal at the Los Angeles General Hospital was given under the supervision of Dr. Ida Heissig, Chief of the Anesthetic Staff.

Table 1 shows the type of operation in which sodium amytal was used.

TABLE I.

Operation		No. of Cases
Thyroidectomy		101
Cholecystectomy		20
Cholecystectomy and Choledocholithotomy		4
Gastro-enterostomy		15
Gastrectomy for Carcinoma		7
Iliocolostomy		2
Carcinoma of the Cecum	1	
Carcinoma of Ascending Colon	1	
Colectomy		3
Descending Colon for Carcinoma	2	
Carcinoma of Sigmoid	1	
Removal of Rectum for Carcinoma		2
Carcinoma of Liver, Exploratory		3
Appendectomy		14
Hernioplasties		10
Inguinal	6	
Umbilical	2	
Ventral	2	
Closure of Cecal Fistula		1
Mastectomy		6
Salpingectomy		2
Hysterectomy		7
Oophorectomy, for Cyst		1
Splenectomy, for Splenic Anemia		1
Removal of Submaxillary gland for Carcinoma.		1
Total		200

PREANESTHETIC HYPNOSIS

The chief object of using any preanesthetic hypnotic is to relieve the fear most patients have of an operation and of taking an anesthetic. Most patients worry more about the anesthetic than they do about the operation itself. Sodium amytal relieves the patient of this preoperative fear, diminishes the amount of anesthetic used, decreases that postoperative nausea and vomiting and allows the patient several hours of postoperative sleep. Patients who have had two operations, one without the sodium amytal and the other with sodium amytal, are unanimous in their praises of this new method.

Dosage and administration according to Zerfas[2]:

When sodium amytal was first used to produce anesthesia, doses as large as 30 grains were given intravenously, and even

then at times ether had to be used. The long postoperative narcosis of twelve to twenty-four hours was an objectionable feature, being responsible for many of the postoperative lung complications, such as bronchopneumonia and pulmonary edema.

In our series no attempt was made to produce anesthesia with sodium amytal, so that the dose we use has been much smaller.

Although there is a rough relationship between the dose and the weight of the patient there are other factors more important such as:

(1) The age and general condition of the patient. Very elderly and debilitated patients require less sodium amytal.

(2) Toxic patients with an elevated basal metabolic rate will require larger doses. Several toxic goiter patients required 22½ grains to produce sleep.

The method of using sodium amytal has been as follows: If indicated, the patient is given 1/6 grain of morphine, 1/150 grain of atropin, about three quarters of an hour before injecting the sodium amytal. The solution is made fresh for each case. It is given with the patient in bed in his room. The blood pressure is taken just before injecting the sodium amytal and retaken after every 3 c.c. (4½ grains) of the solution is injected. The solution is injected very slowly, never faster than 1 c.c. a minute and as soon as the patient is asleep, or if blood pressure drops too rapidly, the injection is stopped. We have no preconceived idea as to the dose necessary but are content to produce sleep, as we always use an anesthetic. Some patients go soundly to sleep on 3½ grains and others, as in toxic goiter cases, have received 22½ grains. The average dose in this series was 9 grains. Usually, in from three to six minutes the patient becomes drowsy and falls into a profound sleep.

The one giving the injection should be familiar with the patient's condition and also familiar with the possible reactions. The sum total of the effects are obtained almost instantly so that there is not much danger of a delayed accumulative action.

BLOOD PRESSURE, PULSE and RESPIRATORY CHANGES.

The most interesting change in the patient's condition is the fall in blood pressure, the greater fall being in the systolic pressure, with very little change in the diastolic pressure. Fourteen cases showed no change in blood pressure and in one case the systolic pressure dropped 96 mm. In several cases there was a drop of 60 mm. The average drop of the systolic pressure was 17 mm., and of the diastolic pressure was 5 mm.

The drop in pressure occurs early during the injection and reaches the maximum in about ten to fifteen minutes and it usually returns rapidly to the former level about the time the patient reaches the operating room.

The following charts, Nos. I, II, III, illustrate some of the blood pressure changes.

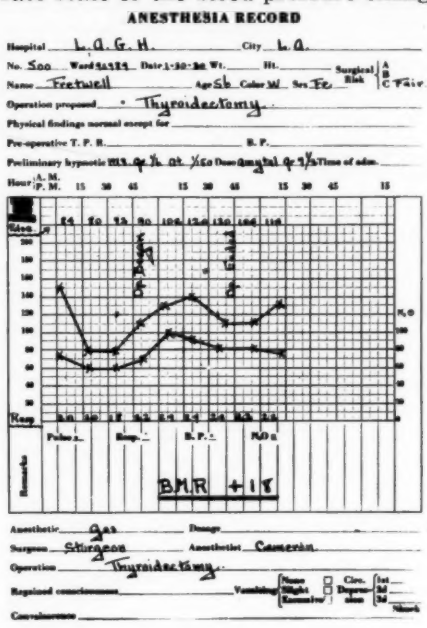

CHART I.

Shows a fall of 60 mm. in the systolic pressure only 10 mm. fall in the diastolic. Very little change in the pulse and temperature.

There does not seem to be a constant relationship between the fall of blood pressure and the amount of sodium amytal used, as illustrated in Table II.

TABLE II.

Operation	Age of Patient	Basal Metabolic Rate	Weight	Blood Pressure Before Sodium Amytal	Amount of Sodium Amytal	Blood Pressure After Injection	Amt. of Blood Pressure Change
Hyperplastic Goiter	47	+27	150	152/68	8 gr.	132/70	-20mm.
Adenoma of Thyroid	44	+12	132	160/70	15 gr.	100/68	-60mm.
Adenoma of Thyroid	48	+19	124	155/62	12 gr.	90/67	-65mm.
Acute Appendicit's	45		210	176/80	10 gr.	80/60	-96mm.
Hyperplastic Goiter	48	+24	176	140/70	22½ gr.	130/70	-10mm.
Gastrectomy	62		120	130/80	8 gr.	80/55	-50mm.
Acute Appendicitis	35		138	110/70	13 gr.	110/60	none
Cholecystectomy	46		168	140/80	15 gr.	138/76	- 2mm.
Acute Appendicitis	35		165	110/75	15 gr.	110/75	none
Cholecystectomy	48		196	160/100	11 gr.	160/95	none
Adenoma of Thyroid	46	+22	140	176/110	22½ gr.	170/100	- 6mm.
Hyperplastic Goiter	22	+ 36	110	140/70	15 gr.	100/60	-40mm.

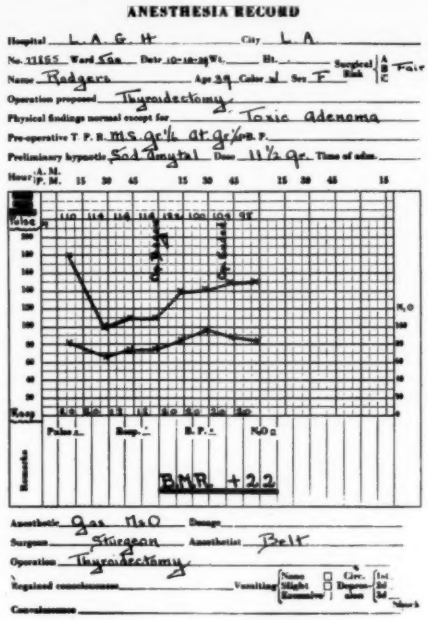
ANESTHESIA RECORD
Hospital L. A. G. H. City L. A.
Name Rodgers
Operation proposed Thyroidectomy
Physical findings normal except for Toxic adenoma
Pre-operative T. P. R. M.S. gr ¼ at gr ½ B. P.
Preliminary hypnotic Sod Amytal Dose 11½ gr. Time of adm.
Op. Began
Op. Ended
BMR +22
Anesthetic Gas N2O Dosage
Surgeon Sturgeon Anesthetist Belt
Operation Thyroidectomy

CHART II.

Shows a fall of 80 mm. in the systolic pressure and only 15 mm. fall in the diastolic. Very little change in the pulse and respirations.

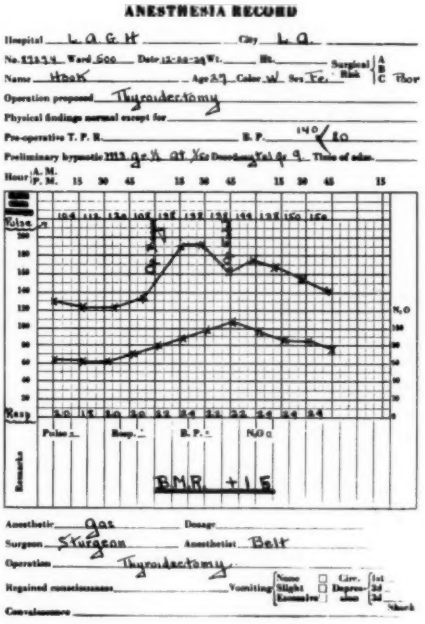
ANESTHESIA RECORD
Hospital L. A. G. H. City L. A.
Name Hoak
Operation proposed Thyroidectomy
Physical findings normal except for
Pre-operative T. P. R. B. P. 140/80
Preliminary hypnotic M.S. gr ¼ at ½ Duoden Sod Amytal gr 9 Time of adm.
Op. Began
Op. Ended
BMR +15
Anesthetic Gas Dosage
Surgeon Sturgeon Anesthetist Belt
Operation Thyroidectomy

CHART III.

Shows a fall of 10 mm. in the systolic pressure, then a rise of 70 mm. Diastolic no fall, but a rise of 30 mm. Pulse varied from 104 to 150. Respirations little affected.

Having had a rather large number of cases in which the fall of blood pressure was rather marked, we have tried giving ¾ grain of ephedrin about fifteen minutes before injecting the sodium amytal. This method has been used for the last seventy-five cases and the results have been very satisfactory. The drop in pressure has varied from none to 30 mm.

The following charts illustrate the slight change in the blood pressure after using ¾ grain of ephedrin.

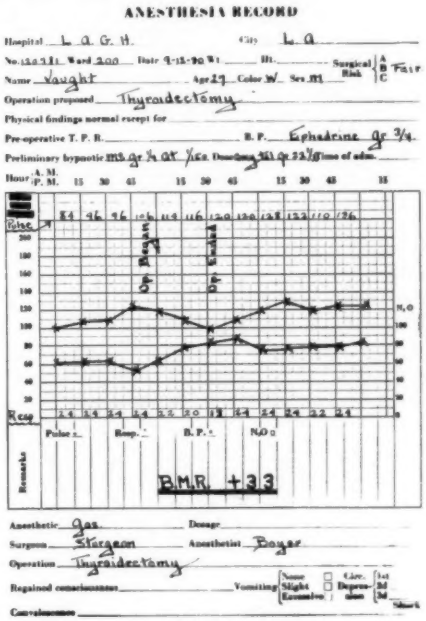
ANESTHESIA RECORD
Hospital L. A. G. H. City L. A.
Name Vaught
Operation proposed Thyroidectomy
Physical findings normal except for
Pre-operative T. P. R. B. P. Ephedrine gr ¾
Preliminary hypnotic M.S. gr ¼ at ½ Duoden Sod Amytal gr 22½ Time of adm.
Op. Began
Op. Ended
BMR +33
Anesthetic Gas Dosage
Surgeon Sturgeon Anesthetist Boyer
Operation Thyroidectomy

CHART IV.

Shows a rise of 10 mm. in the systolic pressure. No change in the diastolic pressure. Very little change in the pulse and respirations.

Many cases showed an increase in the blood pressure at the end of the injection, as illustrated in Table III—all received ¾ grain of ephedrin.

TABLE III.

Operation	Age of Patient	Basal Metabolic Rate	Weight	Blood Pressure Before Sodium Amytal	Amount of Sodium Amytal	Blood Pressure After Injection	Amt. of Blood Pressure Change
Adenoma of Thyroid	47	+24	138	150/105	15 gr.	174/110	+24mm.
Cholecystectomy	41		165	154/100	15 gr.	170/105	+16mm.
Hyperplastic Goiter	24	+28	98	110/80	15 gr.	120/80	+10mm.
Hyperplastic Goiter	19	+33	108	114/74	13 gr.	120/80	+ 6mm.
Obstructive Jaundice due to Common Duct Stone	54		158	112/70	15 gr.	144/80	+16mm.

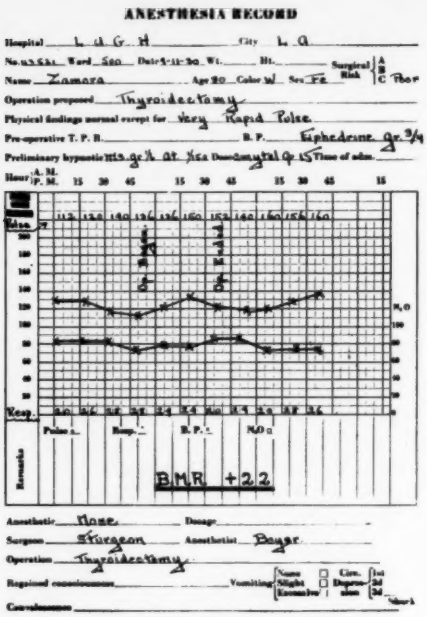

CHART V.

Shows a fall of 10 mm. in the systolic and diastolic pressure. Respirations and pulse very little change.

There has been practically no effect on the pulse rate, as shown in Charts I, II, III, IV and V. The respirations are regular, usually increased. Slight cyanosis has occurred in a few cases.

Supplementary anesthetic agents, as nitrous oxide and oxygen, ether, local and spinal, were used, as follows:

Anesthetic	No. of Cases
Nitrous oxide and oxygen	142
Nitrous oxide, oxygen plus ether	15
Ether	20
Local	10
Spinal	8
No anesthetic	5

Patients under the influence of sodium amytal take the anesthetic much more easily. There is no excitement, no laryngospasm, and the amount of the anesthetic used is much less.

Our preference is for nitrous oxide and oxygen. More oxygen can be used. There is less cyanosis and the muscular relaxation is sufficient in most cases to do any upper abdominal operation.

Although spinal combines very will with sodium amytal, one must remember that spinal anesthetic also depresses the blood pressure. We make sure before giving spinal that the blood pressure has returned to the original level. If not, we give ¾ grain of ephedrin and, if the blood pressure remains low, spinal is not used.

DURATION OF EFFECT

The postoperative sleep lasts from one to eight hours, depending on the dose used. One patient, age 52, with carcinoma of the rectum, slept thirty-two hours on 14 grains of sodium amytal. There was only 5 mm. drop in the blood pressure and the operation was completed without any supplementary anesthetic.

Just before awaking the patients may be slightly restless, but not violent. Usually, a ¼ grain of morphine sulphate will quiet the patient. Patients with hyperthyroidism are more restless than non-toxic cases and require more opiates. About 5 per cent of our patients were extremely restless and one patient was violent.

Postoperative nausea and vomiting are extremely rare, certainly much less than without the use of the sodium amytal.

The administration of fluids both intravenously and subcutaneously is easier while the patient is asleep.

ADVANTAGES

1. The patient is relieved of all preoperative fear.
2. Patient is unaware that the anesthetic is being given.
3. Quiet respirations, making upper abdominal surgery easier.
4. In most cases less of the anesthetic agent is required.
5. Postoperative sleep, with loss of memory for painful events.
6. Absence of postoperative nausea and vomiting in most cases.
7. Also valuable when complications such as hiccups, psychosis, severe pain, idiosyncrasy to opiates, morphine addiction, are present.

DISADVANTAGES AND COMPLICATIONS

1. Moderate degree of hypertension and of hypotension are not contraindication to the use of sodium amytal.
2. In elderly patients with arteriosclerosis and fixed hypertension, sodium amytal should not be used.
3. In patients with badly damaged hearts and hypertension, sodium amytal should not be used.
4. Patients with lung infections, lung abscess or bronchiestasis, only small doses of sodium amytal should be used in order not to interfere with the cough reflex.
5. Persons who are known to be sensitive to any of the barbiturates should not be given sodium amytal.
6. Bronchopneumonia and edema of the lungs are commonly reported.

In this series we have only one case of bronchopneumonia and no cases of pulmon-

ary edema. Both complications are probably due to too large a dose of sodium amytal, with too long postoperative sleep.

CONCLUSION

Any drug as powerful as sodium amytal that can put a patient to sleep within five minutes and that cannot be immediately removed from the circulation, demands great care in its administration. It is only by the best cooperation between the surgeon and the anesthetist that this can be accomplished.

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A REVIEW OF THE PRESENT DAY SURGICAL TREATMENT OF CERTAIN DISEASES OF THE CHEST

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(Read before the Annual Meeting of the Medical and Surgical Association of the Southwest, at El Paso, Texas, Nov. 6-8, 1930).

The fact that any particular surgical procedure is new by no means proves that it is an advance over previous operations performed. Wave upon wave of surgical enthusiasm has swept over the world following extravagant statistics, too soon compiled, of new and remarkable operative cures obtained. In the wake of these waves so many times have been unnecessary suffering, chronic invalidism or death. A conservative discussion from time to time of present-day methods in this still adolescent specialty of chest surgery, is well worth while. If this paper promotes such a discussion it will, in the opinion of its author, have served its purpose well, indeed.

The speaker has but recently returned from a tour of inspection through the principal chest-surgery clinics and sanatoria of Europe. The observations which will be made upon the various diseases of the chest are based, therefore, on both the present-day American and European procedures.

TUBERCULOSIS OF THE LUNGS

From 20 to 30 per cent of the residents in the principal sanatoria of Europe today are under artificial pneumothorax treatment. In the Schatzalp Sanatorium at Davos, in Switzerland, 30 to 40 per cent receive such collapse therapy. There is growing tendency in the European sanatoria, as well as here, to begin pneumothorax comparatively early in the course of an essentially unilateral exudative tuberculosis. There exists the same uncertainty abroad, as here, in regard to the length of time that should elapse before a lung, compressed by pneumothorax, may be allowed with safety to re-expand. It still remains a problem to be settled according to the judgment and experience of each physician. When lung re-expansion is decided upon, permanent phrenic interruption is of definite value, not only in reducing the extent of pulmonary re-expansion, but also in lessening the perpendicular pull following adherence of the lung to the diaphragm. Phrenicectomy may benefit greatly when, under artificial pneumothorax, pleural adhesions to the diaphragm definitely prevent satisfactory lung collapse. The Germans report particularly good results following this procedure and the speaker is thoroughly convinced of its value.

PNEUMOLYSIS

Conservatism in the presence of pleural adhesions interfering with lung collapse during artificial pneumothorax, is becoming steadily more apparent. Unverricht is doing one operation for the division of adhesions now, where he did ten five years ago. If a cavity is situated immediately beneath an adhesion which prevents collapse of its walls, division of this adhesion is not without risk. Cavity rupture, even two to three months after adhesion division, not rarely follows. If there are multiple adhesions with one or more of considerable size, their division is usually inadvisable. The longer and thinner the adhesions, the better the prognosis for relief after their division. Pneumolysis should not be attempted during the first three or four months after the beginning of artificial pneumothorax since pleural resistance to bacterial invasion is notably less during this period. Moreover, many pleural bands that at first resist, often give way under the constant intrapleural pressure. In brief, if the collapse of the lung is unsatisfactory after several months of pneumothorax, and if there are one or more comparatively small adhesions which, divided, might allow the lung to be satisfactorily collapsed, and if there is no cavity within the immediate vicinity of these adhesions, intrapleural pneumolysis is definitely indicated. If, however, the case does not meet these requirements, thoracoplasty often is attended with less risk and more prompt permanent relief.

The open method for the division of pleural adhesions is almost universally condemned. The great majority of patients so treated develop tuberculous pleural effusions, many of which sooner or later become secondarily infected.

PHRENIC INTERRUPTION

There is no question but that this is one of the most abused procedures pertaining to chest surgery today. It is extremely unfortunate that bad results following any given method of treatment in so chronic a disease as pulmonary tuberculosis, should appear, as a rule, so slowly that without careful scrutiny the causative factor is often overlooked. It is this unfortunate fact that hides from us many thoroughly bad results following phrenic interruption. The more conservative men, experienced in both medical and surgical treatment of tuberculosis, rarely, if ever, advise phrenicectomy in the presence of an acute exudative tuberculosis, even though this process is relatively well limited to a lower lobe. Dumerest, in checking over at Hautville cases of progressing exudative tuberculosis where phrenicectomy had been performed, found that, of those operated upon three or more years before, none were alive. His conclusion was that the phrenic interruption had not necessarily contributed to their deaths but that it certainly had not been successful in checking the advance of the tuberculosis.

In chronic productive tuberculosis the situation is an entirely different one. Manifestly, fibrosis and chronicity indicate in a patient more than usual resistance to the tubercle bacillus. In such a situation anything that may contribute to a lessening of the mechanical disadvantages interfering with the obliteration of active tuberculous areas, or to a lessening of the mobility of these parts, is indicated if the patient is not steadily improving, provided the procedure under consideration is warranted by the probable amount of good that it will do. In the speaker's opinion, the routine institution of phrenic exeresis immediately preceding early thoracoplasty, is neither indicated nor advisable. If, under the flouroscope, little or no motion of the diaphragm can be seen and there is extensive fibrosis throughout a lower lobe, little change in the position of this diaphragm can be expected by its paralysis. On the other hand, if the lower lobe shows no tuberculous activity and is comparatively free of markings suggesting old healed tuberculosis, phrenic exeresis succeeds only in throwing out of commission to a greater or less extent that portion of the lung which the patient could use following thoracoplasty. Moreover, the vital capacity in the tuberculous patient is from 20 to 40 per cent less than normal. Phrenic interruption reduces the vital capacity still more and this reduction persists from two to three months just at a time when the need for maximum efficiency in gaseous interchange and cardio-respiratory function is most necessary. If, on the other hand, there is definite diaphragmatic mobility with respiration, and a careful study of the x-ray films, plus the physical findings, leads to the conclusion that a preliminary phrenic exeresis is indicated, the patient should be allowed several weeks to recover his cardio-respiratory equilibrium before thoracoplasty is begun.

In lower lobe tuberculosis of the productive type, phrenic interruption has given dramatically good results. Comparatively small cavities scattered through the lung in this type of tuberculosis occasionally disappear with astonishing rapidity following phrenic exeresis. In large cavities with extensive pulmonary fibrosis, it is seldom that phrenic interrupution, as an isolated procedure, is of great value.

The belief is quite general that expectoration is attended with less effort following phrenicectomy. This is interpreted as meaning that coughing is more efficient. I am convinced that, after the first two or three weeks following phrenic interruption, coughing is not made more efficient by this procedure. In fact, I feel that thorough emptying of the lower lobe bronchi is rendered more difficult. It is amazing, during bronchoscopic examination following phrenic interruption, to see how comparatively immobile is the lower lobe even during the violent coughing attacks that may occur during bronchoscopy. That patients suffering from pulmonary tuberculosis are definitely benefited by phrenic interruption, is most certainly true, but this benefit is probably due to decreased mobility of the affected lung rather than to increased ease in purulent evacuation. Coughing efficiency within the lobe itself is probably decreased. The ciliary action within the bronchi and the peristaltic movements of their walls, unquestionably are of the greatest aid in removing purulent material. It is very probable that, without these two factors, preenicectomy would frequently produce disastrous results. Repeatedly I have produced temporary diaphragmatic paralysis in the treatment of a non-tuberculous lung abscess. There has usually been improvement for two or more weeks, then the condition slowly has grown worse. The lung involvement has extended. I believe that diaphragmatic paralysis decreases lung mobility but does not aid bronchial drainage.

EXTRA PLEURAL THORACOPLASTY

(In Tuberculosis). The selection of cases for this procedure is becoming more and more careful. The rule may be laid down that, with rare exceptions, thoracoplasty does not arrest advancing tuberculosis. Theoretically it would seem that thoracoplasty should accomplish in exudative tubreculosis the amazingly good results that artificial pneumothorax does. This is not borne out in fact. Given a patient with tuberculosis, whose inherent resistance is not great enough to stop the advance of the tubercle bacilli, and thoracoplasty is definitely contraindicated for arresting the progress of the tuberculosis itself. Where, however, there are mechanical difficulties due to adherence of scar tissue to surrounding comparatively immobile structures, the body may be unable on account of these mechanical difficulties to quiet the tuberculous activity. In such a situation, relaxation of the chest wall, graded intelligently both as to the extent of the rib resection and the number of ribs to be removed at each procedure, overcomes this mechanical barrier that has prevented arrest of the tuberculosis and allows nature once more to resume its fight against the tubercle bacilli without handicaps. If the past history of the patient discloses a resistance which was able at first to control the advance of the tuberculosis, the prognosis in such cases is very good. If thoracoplasty is performed on a patient who has never shown definite tissue reaction to the tuberculous invasion, the results, in my opinion, are almost invariably bad.

The three cardinal points for consideration when thoracoplasty is contemplated in a given patient, are: first, his past and present resistance to tuberculosis; second, the mechanical situation that exists and how much, if any, relief of this can be accomplished by thoracoplasty; third, how great a load, from the patient's history, the heart has had to carry and how well able it is to meet the emergency of the drastic and sudden physiological change occurring with multiple rib resection.

The optimum time, certainly, for thoracoplasty is during the first five years of suppuration. After ten years of chronic toxicity, and the patient's general resistance is greatly decreased and his cardiac reserve is very low indeed. There can be no definite age limit established. A man of thirty-five with a history of eight or ten years of chronic pulmonary tuberculosis is a poor risk for thoracoplasty. The risk is increased if his life has been a particularly inactive one. A man of forty-five, on the other hand, whose general condition is comparatively good, whose resistance to tuberculosis has been above the average, and whose history of pulmonary suppuration is within the five-year limit, may well go through thoracoplasty with splendid results.

MULTIPLE RIB RESECTION IN LUNG ABSCESS

In the early days of non-tuberculous lung abscess, complete rest and medical supportive measures are indicated. The external drainage of an abscess in its acute stage ends in disaster. Surrounding an area of necrosis within the lung is a zone of pulmonary lobules choked with pus. Gradually, as the acuteness of the infection subsides, this pus within the surrounding lobules is expectorated, leaving a fairly well-defined abscess area. It is essential therefore that the coughing efficiency be not decreased. When an abscess is drained externally many bronchi are necessarily opened. Air, with each respiratory cycle, instead of contributing to the efficiency of the cough and producing expectoration, rushes back and forth through these bronchial fistulae. The com-

pression, which is so essential to bronchopulmonary drainage, is lost. Purulent material in the surrounding lobules can not, therefore, be expectorated and necrosis in these tissues follows. Thus the vicious circle is complete and death from advancing pulmonary suppuration follows. Until the area of lobular suppuration surrounding an abscess is evacuated of its pus by expectoration, external drainage of a lung abscess may not be considered.

When, during the acute stage, an abscess has ruptured into a bronchus and purulent expectoration exists, postural drainage is of very great help. During postural drainage, however, when the patient subjects himself to most violent spasms of coughing there is considerable danger of hemorrhage. It is the rule, following a severe hemorrhage, for blood containing bacteria to lodge in contiguous or distant bronchiles, from which secondary abscesses often develop. This complication is a disastrous one. Postures producing such violent spasms of coughing should therefore be avoided. Often, bronchoscopic catheterization of the main bronchus draining an abscess, makes drainage much more free and contributes definitely to healing. If after six or eight weeks the patient is not showing steady improvement, atrificial pneumothorax very carefully given with repeated but small amounts of air, may contribute largely towards abscess wall collapse. Artificial pneumothorax should never be undertaken, however, unless one is convinced that the abscess lies deep within the lung. If, during this treatment, x-rays show adhesions to the chest wall, pneumothorax should be at once discontinued on account of the danger of rupture into the general pleural space. When the above measures have failed to produce improvement, it is customary to consider external drainage. The procedure is done in two stages: first, a local thoracoplasty at the point on the chest wall nearest the abscess; then, as a second step, wide cauterization into the abscess itself. Even the most favorable cases after this procedure drain for many months. The danger of septic cerebral emboli is a very definite one. Many cases never entirely heal. In multiple abscesses this procedure is notoriously ineffective. External drainage, however, has reduced the mortality nearly fifty per cent in comparison with continued medical treatment.

The writer has developed a new treatment for subacute and chronic lung abscess, which, instead of interfering, as external drainage does, with nature's method of repairing the lung following local necrosis, is meant rather to aid nature in overcoming the mechanical barriers which are porhibiting repair. In pulmonary suppuration, whether it be acute or chronic, the body depends for drainage upon the bronchi. When the patient's resistance is good and bronchial drainage is free, the majority recover spontaneously. It is usually the failure of the abscess walls to collapse that calls so urgently for surgical relief. The procedure is, briefly, as follows: Ribs are resected somewhat widely in the region of the lung abscess and, by slow and gradual compression through gauze packing, the abscess wall, which by adherence to the surrounding structures has prevented healing, is relaxed. With this hindrance removed, the lung is able once more to proceed with its own efficient method of broncial drainage and repair. While it is too soon to report the cases treated in this manner, yet in the comparatively limited series on which it has been tried the results so far have been very gratifying. It is amazing how a comparatively limited removal of chest wall support will aid lung healing. We have found that rib resection lateral to and somewhat above the site of the lung necrosis seems to be more effective than sudden and marked relaxation directly over the abscess itself. The chest wall is strapped tightly in order to preserve intrathoracic compression for bronchial drainage. It is essential that the abscess wall be very gradually relaxed in order to avoid as far as possible the occlusion of bronchi draining the abscess.

TUBERCULOUS EMPYEMA

This is truly one of the most difficult problems confronting those who treat diseases of the chest. Opinions in regard to treatment have been, up to the recent past, nearly as numerous as the doctors who treat them. Gradually, out of the chaos of conflicting opinions, rational effectual treatment is developing.

Pleural effusions occurring during the course of pulmonary tuberculosis may be divided roughly into three types:

(1) Serous pleural effusions containing few blood cells and no tubercle bacilli, occurring with or without artificial pneumothorax. The consensus of opinion today is that obliteration of the pleural space is not indicated unless thoracoplasty is held to be the procedure of choice for the underlying lung. It is important, of course, that the character of the pleural fluid be watched.

(2) Sero-purulent effusions rich in lymphocytes and containing tubercle bacilli but no secondary pyogenic organisms. If this effusion persists, despite frequent aspirations, for several weeks, and if the fluid becomes more purulent and thicker, the pre-

vailing opinion in Europe today and the growing conviction of many in North America, is that thoracoplasty is usually advisable. It is during the course of lung collapse by artificial pneumothorax that this type of effusion usually develops and a thoracoplasty, therefore, is but the substitution of one procedure for another to achieve the same end. It is true that various dyes, iodine solutions, formalin in glycerine, and so forth, have been used with eventual obliteration of the pleural space. The danger of secondary infection during this time is always a serious menace, however. Even though the obliteration of the pleural space may be accomplished it is by no means rare to find that the mediastinal displacement to the affected side is so marked that serious circulatory disturbances develop. The risk of a two-stage or three-stage thoracoplasty is very little since the lung is already collapsed. The constant menace of secondary infection while the tuberculous empyema exists, is a considerable one.

(3) Tuberculous empyema with acute secondary infection. Few complications are more serious in the course of pulmonary tuberculosis than this one. The physiologic demoralization of the patient is extremely rapid. The death rate is terribly high. The initial treatment of this condition is conservative. Aspirations with the immediate introduction of one of several dyes, of Rivinol of Jesson's Iodine Solution, or of formalin in glycerin, is the first procedure. The response to this treatment depends upon the extent of the secondary infection. If the inflammation is comparatively well limited to the pleural surfaces, even though absorption through the lymphatics be considerable. relatively complete aspiration of the purulent material and thorough lavage with some mild antiseptic usually produces improvement within a few days. If the source of the secondary infection is eliminated, the secondary organisms may disappear entirely. Unfortunately, in the majority of cases the infection extends everywhere throughout the thickness of the pleura and even may activate the subjacent lung tissue. When the patient becomes steadily and alarmingly worse under pleural lavage, it should be assumed that the latter condition exists. Ultra-conservative measures lead then only to disaster. The Europeans do a pleurotomy with or without rib resection without further delay, as a rule trying to maintain air-tight drainage. Pleural lavages are frequent, though it is generally acknowledged that by this method but a small portion of the empyemal walls is reached by the solution. Usually the patient's condition has become so grave that collapse therapy is precluded. He must be improved before surgery may be considered. It is during this time that the mortality is so great. The writer is becoming more and more of the opinion, since, in an overwhelmingly majority of cases, lung re-expansion can never occur in this grave type of tuberculous secondarily-infected pan-pleuritis, that wide-open drainage of this collection of pus should be instituted while the patient's general condition is comparatively good, in order, as far as possible, to maintain it so. Gauze saturated with some of the several antiseptic solutions, packed snugly against both the parietal and visceral pleura, will come in intimate contact with all parts of the infected cavity. Archibald of Montreal, is the only one so far who (to my knowledge) has remonstrated against these prolonged conservitave measures. If the patient's resistance permits him to survive these early days, he gradually begins to regain his strength, and then, at the earliest possible moment, graded extensive thoracoplasty is performed, to bring in apposition the parietal and visceral pleurae. This procedure is the logical one. It is surprising in what a large percentage of cases healing occurs with or without a small draining fistula, despite the fact that the process is basically tuberculous. The earlier the pleural walls can be brought together, the more assured we can be of satisfactory healing. The point for emphasis, therefore, in the treatment of this latter class is that free and dependent drainage should be, if not at once accomplished, at least not delayed until the patient is nearly moribund. While it is surely granted that the patient's inherent resistance is of the greatest importance, the proper time for the establishment of free drainage may, nevertheless, decide his fate.

BRONCHIECTASIS

It is probable that many times this sort of chronic bronchial suppuration occurs in patients who were born possessed of markedly dilated bronchi. No symptoms develop referable to them, however, until some acute respiratory disease produces inflammation in their walls. How often following whooping cough, measles, or pneumonia, a persistent productive cough proves to be due to a bronchiectasis, the etiology of which cannot be satisfactorily explained by the recent acute disease alone!

Rest, careful attention to cranial sinuses and the relief of upper respiratory infections, plus postural drainage have been the routine procedures which have proved the most effectual. Very recently Ochsner has reported a considerable series of cases treat-

ed by intrabronchial introduction of lipiodol. Twenty-eight of the cases so treated were relieved of their symptoms. The secret of his success is due, apparently, to his thorough anesthetization. Thorough cocainization of the fauces and anterior pillars, including the tissues on either side of the base of the tongue, renders the patient temporarily unable to swallow. The lipiodol is then poured down over the base of the tougue. In this way pus in the dilated bronchi of the lower lobe is, in a very great measure, replaced by the lipiodol. This thorough replacement is apparently responsible for his splendid results. It is a well-established fact that in bronchiectasis involving both lower lobes, if there is relatively little involvement on one side, local measures bringing about improvement on the side showing the greater involvement produce often marked improvement in the contralateral side. Artificial pneumothorax, therefore, on the side of the more extensive involvement, not infrequently is of great benefit whether there is minor involvement of the other side or none at all. If the lower lobe is the seat of the bronchiectatic involvement and it is found that it does not pull free in its mesial half from the diaphragm, phrenic interruption occasionally greatly aids during artificial pneumothorax treatments. If the bronchiectatic involvement is extensive but limited to a lower lobe, and if the measures enumerated above have not been successful, lobectomy certainly is the method of choice. Bronchiectasis is progressive. The prognosis for continued self-support for the patient is definitely limited. There are at best but a few years of even comparative comfort left to the patient. An operation, which in experienced hands has a mortality of fifteen per cent or less, represents one of the recent outstanding advances in the treatment of this dsiease.

The principles of the procedure are thoroughly sound and are, in brief, as follows: It is essentially a two-stage operation. At the first stage the 7th, 8th and 9th ribs on the affected side are resected from the spinous processes to the mid or anterior axillary line. The pleura is then widely opened. The lung within collapses. The lower lobe is gently freed from the lobe above and from the diaphragm. If this cannot be thoroughly done this type of lobectomy must be abandoned. If one is successful, however, the lobe is freed to the hilum. The pulmonary ligament is partially divided and stripped upward. Following Alexander's suggestion, all the pleural surfaces, not only of the lower lobe but of the lobe or lobes above, and the interlobar fissures are rubbed with dry gauze. This irrigation is sufficient, with re-expansion of the lung, to cause adhesions of the visceral pleura to the chest wall. The incision in the chest wall is then closed tightly. At the second stage, some six or eight days later, the original incision is opened. The lung is found uniformly adherent. The lower lobe is carefully freed from the chest wall, the diaphragm, and the lobe above. The latter remains adherent to the chest wall and does not collapse. Infection of the general pleural cavity is thus avoided. The hilum of the lower lobe is easily exposed, doubly ligated, and these ligatures in turn are fastened to the soft spots of the chest wall. The lower lobe, which by this time has shrunk to approximately one fourth of its original size and has become black, is left to slough off exteriorly on the chest wall. The wound is tightly closed about the stump. The local pleural infection following this is controlled by a stab wound at the costo-phrenic angle. In the writer's opinion, as experience in the technic in this operation increases, the mortality in this type of lobectomy will be reduced within a comparatively short time to ten per cent or less.

Lobectomy as performed in Sauerbruch's clinic and in Zaaijer's, is also carried out in two stages. The procedures in principle are essentially the same as ours. In place of immediate stump ligation, however, a rubber catheter under tension is anchored tightly about the lobe hilum and by steady contraction gradually produces strangulation of the lobe distal to it, with eventual amputation. Their end results vary little from ours.

For bronchiectasis limited to a lower lobe, or for early carcinoma within the lobe bronchus, it is of outstanding value, and when no other procedure offers hope, lobectomy should certainly be considered. If artificial pneumothorax is tried and proved unsuccessful and lobectomy is decided upon, the lung must not be allowed to re-expand until the time of operation, since the pleural adhesions following lung re-expansion may prevent subsequent freeing of the lobe for removal and so preclude this comparatively safe type of lobectomy.

In the event that it is found impossible within the limits of safety to free the lower lobe for excision, Graham's cautery pneumectomy is the procedure of choice. By the actual cautery, after extensive rib resection, the dilated bronchi filled with pus are burned across so that the purulent material, by gravity, empties itself into the wound. This operation often gives splendid results. The mortality, however, is approximately twenty-two per cent. One hesitates, there-

fore, to advise this procedure strongly to a person still in comparatively good health. When nothing less radical can be offered him, however, and he realizes that the prognosis is black indeed without operation, the risk is worth the taking.

Indications and contra-indications have been stressed in this paper, for they constitute the frame work which, when properly arranged, makes the finished structure secure. Cerebration is infinitely more important than manipulation in producing cures in diseases of the chest. With a broad knowledge of thoracic physiology and anatomy, chest surgery per se is not more difficult than major surgery elsewhere in the body. To the man who knows how much to do and when to do it, comes the most success, even in chest surgery.

TRACHEOBRONCHAL LYMPH NODE TUBERCULOSIS

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(Read before the El Paso County Medical Society, October, 1930.)

The subject which I have to present to you tonight is one which has, I believe, a profound interest for all; namely, the character, pathogenesis and clinical course of childhood tuberculosis.

The title "Tracheobronchial Lymph Node Tuberculosis" was selected in the effort to stress the importance of the infection of these glands in infancy and childhood. The subject will be taken up by a discussion. first, of the anatomical pathology involved; second, of the experimental work brought forth in recent years, especially emphasizing the differences between the primary and the secondary infections of tuberculosis and stressing the extremely acute character of the primary infection as opposed to the chronic; and lastly, cases illustrating the points made, will be cited.

Dr. Opie, professor of pathology in the University of Pennsylvania, and director of laboratories of the Henry Phipps Institute, has well stated, "Tuberculosis of early life has the characters of a primary infection; whereas, tuberculosis of later life is a disease modified by acquired immunity."

Parrott, as early as 1876, stated, "There is no affection of the lungs which is not also present in the adjoining lymph glands, and vice versa; no changes occur in the peribronchial lymph glands without analogous ones in the lungs."

Cornet, of France, has shown that regional lymph glands enlarge at the point where infection occurs.

Albrecht, in 1906, pointed out that one can usually discover a gross pulmonary lesion corresponding exactly to the regional lymph nodes involved.

Nagaeli, likewise, stated, "That only a pulmonary lesion could be assigned to a tuberculosis of the bronchial glands; the primary focus being in the lungs themselves." This, therefore, shows the inter-relationship between primary pulmonic lesions and adjacent lymph node involvement.

Dr. Abt has classified bronchial gland tuberculosis under the following heads:

(1) Tracheobronchial lymph node involvement.

(2) Bifurcation lymph node involvement.

(3) Hilar gland involvement, the last being sub-divided into the two divisions of bronchial pulmonary lymph node involvement and pulmonary lymph node involvement.

It may be stated that Abt points out that the glands of the right side are usually more involved than those of the left.

Funk, clinical professor of medicine at Jefferson Medical College, and a contributor to "Oxford Monographic Medicine," states, "In early childhood, the primary focus occurs in any part, not infrequently the pleura, while the adjacent lymph nodes at the hilum are always affected, sometimes more conspicuously than the primary focus itself." This combination of pulmonary and lymph node involvement, as a result of first infection, is termed the "primary complex" and it has been shown that these primary pulmonary lesions are usually small.

Opie, of St. Louis, following postmortem studies of both children and adults, has found primary lesions in 43 per cent of children of ages 2 to 5 years, in 55 per cent of children of ages 5 to 10; in 65 per cent of children, 10 to 18 years; and in 90 per cent of persons of ages 50 to 70 years. Furthermore, he states, in a very recent article, that results of tuberculin tests done by the Mantoux intradermic method on 4,100 children, show a gradual increase in incidence of reaction, from 47.2 per cent at age of 5 years, to 95.3 per cent at 18 years, which is only moderately above the figures as determined from autopsy examinations.

Furthermore, renewed study of the anatomic pathology of tuberculosis, by means of roentgenologic films prepared from lungs removed after death, has shown that, in persons dying from chronic pulmonary tuberculosis of the adult type, small primary lesions, as shown by calcified scars of healed tuberculosis acquired during childhood, are almost invariably found.

J. Carr, of England, following morbid

pathological studies, has determined that the primary source of infection of cervical lymph glands is usually the tonsils or some portion of the pharyngeal fauces. The mediastinal glands have, as a primary focus, the lungs, and the primary source of infection of the mesenteric nodes is an often inconspicuous break in the alimentary tract. Restated, one may say (1) that in a previously uninfected person infection of the tonsils or fauces by tubercle bacilli induces a cervical adenopathy; (2) primary focal infection induces bronchial gland involvement; (3) primary intestinal tract infection induces the so-called tabes mesenterica.

Abt, in speaking of the pathology of tuberculosis, states, "Tuberculosis in infancy is an acute disease, often ending fatally with miliary lesions that fail to become encapsulated; while tuberculosis in adults is a chronic disease that easily tends to recovery." In this regard, the experimental studies of Opie with immunized and non-immunized guinea pigs, is quite enlightening. In normal animals injected with foreign proteins for the first time, the protein rapidly leaves the site of injection, producing no inflammatory reaction, and enters the circulating blood, where it remains. In animals immunized and sensitized by such an injection, reinjection with the same foreign protein produces intense inflammatory reaction with necrosis at the site of entry.

Opie has shown that foreign protein on second injection is arrested by inflammatory reaction at the site of injection, from which it can be recovered almost quantitatively.

The paradox betwen susceptibility to local inflammation and necrosis, on the one hand, and resistance, that is to say, immunity, on the other hand, is only apparent, for the local inflammatory reaction is the effective means for protection of the internal organs. The similarity between reaction of an animal to a previous foreign protein and the reaction of an animal to a second inoculation with tubercle bacillus, is demonstrated by works of Krause and Willis, who show, by inoculation of excised tuberculous tissue into normal guinea pigs, that there is a rapid dissemination of tubercle bacilli from the site of inoculation, whereas, in immunized animals the infection is remarkably retarded. In the normal animal transit of bacilli from point of entry into the skin to the local lymph nodes 4 or 5 cm. distant, was made in 24 hours or less; in immunized animals, transit for the same distance required from 2 to 3 weeks, and the number of bacilli reaching such nodes was greatly reduced. Thus, from experimental evidences, one can state that inflammatory reaction produced by tuberculosis in a susceptible animal prevents dissemination of the bacteria.

The problem of frequency of pulmonary infection is made somewhat clearer by studies of Lueri, of Henry Phipps Institute. He injected tubercle bacilli intravenously into rabbits, using both human and bovine bacillus, and, by methods developed by him, determined the number of bacilli surviving in any one organ at a given time. He shows that tubercle bacilli are deposited in greatest numbers in the following organs in the order named: (1) the spleen; (2) in diminishing numbers in the liver, lung, marrow and kidney. At first, the bacilli multiplied in all the organs; the human bacillus according to its pathogenicity in vitro more rapidly than the bovine, but, with the bovine type, destruction of these organisms begins after 4 weeks in the liver, spleen, and marrow, but they continue to multiply in the lung.

With the human bacillus, multiplication in the lungs is far greater than in other organs, and destruction proceeds more slowly.

In immunized animals destruction of bacilli follows inoculation almost immediately, although bacilli persist in the lungs in large numbers for months. This experimental work is in keeping with the story of the pathology of infection in the bronchial lymph nodes as found in infancy and childhood.

Garrod, Batton, Thursfield and Patterson, pediatricians of England, in writing of intrathoracic tuberculosis, speak of infection by inhalation to a small focus, where primary infection starts with extension from this primary focus to the adjacent lymph glands. There one of two processes ensues: (1) caseation, fibrosis, calcification and healing of the lymph nodes, or (2) caseation with subsequent rupture of lymph node contents into the trachea, bronchus, pleura, esophagus, pericardium or blood vessels, following which, one may get one of the following types of distribution:

(a) Miliary tuberculosis, pulmonary
(b) Miliary generalized tuberculosis
(c) Tuberculous meningitis
(d) Direct spread into the hilar glands and pulmonary glands
(e) Retrograde lymphatic spread causing apical tuberculosis.

It is because of this danger of caseation and rupture of lymph nodes into the bronchus or blood vessels, causing widespread pulmonary or generalized miliary tuberculosis, that the subject of bronchial lymph-

node involvement as the primary childhood type is so important.

That the pulmonary infection is the disease of greatest import is shown by the fact that 90 per cent of all deaths from tuberculosis are intrathoracic in origin, and, as the term "primary complex" includes involvement of the adjacent lymph nodes, we can readily understand the importance of their infection in young children now infected for the first time.

Next, I wish to speak concerning the routes of infection and later of their sources. First, there are no known cases of germinative infection. Second, placental tuberculosis does occur and tearing of such a tuberculous placenta during birth may transmit the organisms to the child; in fact, organisms have been recovered from the blood of the umbilical cord and liver of the newly born. Also, amniotic fluid can be a source, but, by far the majority of infections that natal: The two methods of infection that occur, are naturally post-natal. Third: Post- undoubtedly produce the vast majority of cases are: infection by inhalation and infection by ingestion.

Von Pirquet estimated that 95 per cent of all cases occurred by this respiratory, bronchogenic or aerogenic route of infection. Cornet, Flugge and Ghon all favor the inhalation method; but, on the other hand, Von Behring and Calmette favor the intestinal route, as the primary one. Von Pirquet, Hamburger, Koch and Finklestein give the alimentary tract a second place as a portal of entry. All authorities on sources of inflammation place infection of the alimentary tract as more common after the first year, with infection of the bronchial nodes as the most common during this year of life; but, without much doubt, man as an individual with active tuberculosis is our main source of infection. Nuttall estimates that such a phthisical patient expectorates from one-half billion to four and one-third billion bacteria per 24 hours and Lamson has shown that 67 per cent of individuals, where there has been contact with active tuberculosis, show evidence of infection.

Carr, of England, shows that the bovine tubercle causes one half of the fatalities of abdominal tuberculosis, causes 50 per cent of the cases of cervical adenitis and about the same percentage of cases of lupus; but that bronchial lymph-node involvement is almost invariably due to the human organism. Avian tubercle bacillus is a very mild infection for humans and need not be considered as a pathogenic organism of tuberculosis.

Further evidence that patients harboring the active disease are the greatest source of infection for children or infants, is shown by the following citations.

Reich pointed out that in a certain German city a certain tuberculous midwife made it a practice to suck the mucus from the mouth of the newly born and, in thirteen months, ten infants so delivered died of tuberculous meningitis. Among those delivered by all other midwives, not a single child contracted the disease. Also, three Japanese workers have shown that the tubercle bacillus can be found in the breast milk in ten out of thirteen nursing mothers with cases of incipient tuberculosis, and in the breast milk of all with far-advanced tuberculosis.

Funk pointed out the following order of frequency of involvement of the organs: (1) the lungs, (2) the bronchial glands, (3) the pleura, (4) the spleen and liver, (5) the mesenteric lymph nodes.

Next, the study of resistance and of its factors will be presented. It has been conclusively shown that the primitive races acquire the infant or childhood type of tuberculosis with usually fatal results, while the civilized races have an acquired resistance causing a chronic form of disease with a great tendency to limitation and recovery.

Individually resistance depends on age, sex, previous diseases and social and economic factors. In the first year of life the disease is very acute and has a high mortality. From five to fifteen years, resistance breaks, and it has been shown that, during the so-called productive period of life, the highest mortality occurs.

In children, particularly, measles and whooping cough predispose to the disease. Acute diseases of all sorts activate disease present; and chronic debilitating diseases predispose to it, if it is not present. Other factors of individual resistance depend upon social and economic conditions, including exposure, economic status of the country and industrial depression and epidemics.

Now a short outline of symptoms and physical findings, in lymph node involvement. The symptoms may be either generalized or local, or both.

1. Generalized Symptoms.
   (1) Lassitude.
   (2) Loss of appetite.
   (3) Irregular temperature.
   (4) Anemia and wasting.

II. Local Symptoms.
   Signs of pressure or irritation, such as pain or cough. The cough may be harsh or metallic and catarrhal.

III. Physical examination with enlargement

of the mediastinum lymph nodes, reveals:

(1) Dullness on percussion between the vertebral column and the border of the right scapula.

(2) The Eustace Smith bruit may be heard over the right sterno-clavicular juncture when the head is fully extended.

(3) D'Espine's sign, consisting of bronchial breathing and whispered pectoriloquy over the vertebra below the 3rd thoracic spine.

(4) X-rays are also helpful, showing mediastinal shadows cast by glands about the trachea and bronchus. The so-called hilar type of tuberculosis reveals an advanced type of mediastinal gland involvement.

(5) Besides physical signs and x-rays, must be added one test which, in the hands of recent investigators, has proved of greater value than any other one test; namely the Mantoux intradermic tuberculin test. In fact, I believe that the clinicians today are wont to place a strongly positive Mantoux tuberculin test next in importance to a clinical history; for physical findings are only too few and difficult of ascertainment in bronchial lymph node tuberculosis.

By a few examples, I wish to stress the importance of contact in childhood tuberculosis, and the help in diagnosis that may be obtained from Mantoux and x-ray findings. I wish to thank Dr. Barrett for the following clinical data obtained from his clinic and the help he personally gave in the assembling of it.

LUJAN FAMILY

This family has five children. The child first seen was Luis, aged 5, whose father died of pulmonary tuberculosis in the City-County Hospital in April of this year, and whose young sister died, following the death of her father, with tuberculous meningitis, May, 1930.

Luis had a positive tuberculin test; clinical findings were malaise, glandular enlargement, loss of weight and temperature 99.0 to 99.6.

Physical Findings: Negative, with the exception of malnutrition and temperature. X-ray: Shows bronchial-hilar nodes with peribronchial thickening. This child is considered as a case of latent tuberculosis, and here, may I state that Opie makes a distinction between latent and manifest childhood tuberculosis; he calls those cases latent, where positive clinical findings are present with positive Mantoux and suggestive x-ray findings; while manifest tuberculosis is an active progressive lesion easily recognizable by both clinical and x-ray findings.

Second Child: Enriqueta Lujan, aged 3 years, whose symptoms were loss of weight and poor appetite.

Physical Findings: Negative, except for malnutrition and temperature, 99.0 to 100.6. Mantoux Test: Strongly positive in one to ten thousand dilution. X-ray findings: Enlargement with peri-bronchial thickening. This is considered as a case of latent tuberculosis.

Third Child: Roberto Lujan, aged one month, born after the father's death. The child is undernourished and is a feeder in the Clinic with temperature 98.6 to 100; Mantoux test is negative.

SECOND FAMILY (McGhee).

The second family is that of McGhee, two children. Doris, aged eight, was brought to the Clinic because she was frail, undernourished, pot-bellied and nervous. Her father is still living, but has had tuberculosis for the past nine years.

Examination The child shows generalized glandular enlargement, gives the story of repeated attacks of tonsillitis and fever. Pulmonary examination is completely negative. Mantoux test is strongly positive. X-rays show hilar thickening with marked peribronchial thickening.

Diagnosis: The diagnosis lies between latent and manifest tuberculosis, though the signs are hardly sufficient to warrant the diagnosis of manifest tuberculosis.

The brother, aged 12, presents no symptoms and a negative physcial, but has a strongly marked Mantoux test.

THIRD FAMILY (Blackburns.)

Three children:

(1) Louis, aged 6, is brought in because of contact with his father, who has been at a local sanatorium for treatment of pulmonary tuberculosis. The child is underweight. He appears well nourished, but has a temperature varying from 98 to 99. The Mantoux test is 4 plus. X-rays show hilar changes with peribronchial thickening and calcified nodes.

(2) Clarence, aged three, has negative physical examination, with standard weight; however, his Mantoux test is 4 plus and x-rays are suggestive of hilar involvement.

(3) Charlotte, aged ten, has negative history, negative physical findings; only a questionable Mantoux test and negative x-ray findings.

So, in this family, two out of three children have positive evidence of tuberculous involvement from a tuberculous father.

In the Lujan family, one child has died, one is unaccounted for, and 3 have positive evidences of tuberculous infection, following contact with the father, who died of tuberculosis.

In the McGhee family, both children show positive findings of infection by Mantoux, though, clinically, only one child suffers with tuberculous involvement.

FOURTH FAMILY (Hill).

Three children: ages 3 years, 17 months and 3 months. The aunt has been diagnosed as a case of tonsillar tuberculosis; has been fond of the children and in close contact with them. The mother brought in the oldest child for examination and physical findings. Mantoux test is 4 plus; x-rays show interweaving at apices with soft shadows, presumably tuberculous.

Second child, aged 17 months, appears well nourished; no clinical symptoms, but this child has a 4 plus Mantoux and x-rays plainly show soft mottling.

Third child, aged three months, has negative history, negative Pirquet test and x-ray.

In conclusion, I wish to state that, although measles and whooping cough cause

marked enlargement of bronchial nodes and peribronchial thickening, they are likewise the two most serious predisposing causes of childhood tuberculosis.

Hence, in any case of bronchial lymph node enlargement, the presumptive evidence is that of a tuberculous involvement of the lymph nodes until proved negative.

HEART SYMPTOMS DUE TO THE EMOTIONS

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This paper will be confined to a consideration of cardiac symptoms of emotional and functional origin. In one half of all patients that I have examined because either they or some physician feared organic heart disease, I have been unable to find convincing evidence to support such a diagnosis. A large part of errors are due to wrong interpretation of symptoms. A few examples will illustrate:

A small child is found to have an irregular heart and organic disease is suspected. On examination we find that the heart is rapid on inspiration and slows markedly on expiration. We all know, but do not always remember, that this is sinus arrhythmia due to the vagus and has nothing to do with organic disease.

A woman, aged 43, was seen April 29, 1929. She complained of pain about her heart, aching in her left shoulder, shortness of breath, and indigestion. She said that her heart jumped and seemed to turn over. She had a short dry cough, was extremely nervous, and could not sleep. The heart and general examination was negative. Fluroscopic examination was negative for pathology. During the taking of the electrocardiogram I noticed several premature beats and that the cough immediately followed these beats. As we all know, premature beats are harmless. In this case, no doubt, the overdistended left auricle, during the pause folllowing the premature beat, pressed on sensory nerves and caused the cough. The other symptoms were due to fear. She was given quinidine, 3 grains twice a day. She returned in two weeks very much improved, due mostly, I believe, to the fact that her fears had been dispelled by an explanation of the nature and harmlessness of her symptoms.

A man, aged 47, has for 30 days noticed a sensation like a bubble of gas, starting below the heart and going rather slowly up the left side of the sternum into the neck. There is a sensation of tightness over the stomach and he belches a great deal. He has shortness of breath on going up stairs, but no pain. He fears heart trouble. Neither the history nor the physical examination, neither the x-ray nor electrocardiogram gave us any clue to an explanation.

His doctor had him off tobacco without result. I told him to go home, eat a good meal, smoke three or four cigars and return in three hours. I then had him exercise and, on sitting down, I caught several premature beats. I then tried the electrocardiograph and with every premature beat he would say, "There is the bubble again." His doctor was advised to give him quinidine and, at last reports, he has been much better.

Here we see false cardiacs in the making.

The following case was more fully developed:

Mrs. B., aged 34 years, was seen March 11, 1930, because of pain over the heart, going to the left neck, shoulder, and arm, and also in the back between the shoulders. She had dyspnea on walking, and palpitation, which was attributed to gas on the stomach. She was afraid to eat because it aggravated her symptoms.

She had the Spanish influenza in 1918 and pyelitis during her first pregnancy. Fourteen years ago she had rheumatism and one year ago she had a tonsillectomy. Three years ago a lump was removed from her right breast. With the exception of nervousness and palpitation she has been quite well.

December 13, 1929, her second child was born. Everything ran the usual course until the twelfth day. Then, while lying in a cramped position and nursing the baby, she suddenly felt a sharp pain in her groin, and then a choking sensation, and then she fainted. The nurse said that the pulse jumped suddenly to 160 and the respirations were 32. The heart rate maintained the same rate of 160 for a week, during which time she was very nervous and sleepless. After the attack she developed a phlebitis in the right leg. The attending physician felt that Mrs. B. had suffered embolism of the coronary artery, and informed the family that the situation was very serious. The pain about the heart and other symptoms subsequent to the attack were attributed to cardiac infarct. Naturally she became alarmed and constantly feared heart failure and death.

Examination showed many caps and fillings in the teeth and considerable pyorrhea; acne quite marked; thyroid normal; all heart sounds well produced and of normal intensity; no murmurs; an occasional premature beat. Arch 5 cm. wide; heart border 8.5 cm. to left and 3 cm. to right. No edema in lungs or elsewhere, no cyanosis. Liver not palpable; some tenderness to pressure over gallbladder. Pain goes upward. Chest tender over second and third left ribs arteriorly. X-ray of heart showed it normal in size and shape. Graham test gave marked delay in emptying of the gallbladder, which appeared atonic and suggested old cholecystitis. X-ray of teeth negative for abscesses. There was marked general alveolar absorption. The blood count was normal. Hemoglobin, 85 per cent; basal metabolism, 7 per cent. Electrocardiogram was normal. Wassermann negative.

Discussion: This rather extensive examination was made in order to settle definitely the question of heart disease. When a patient comes convinced that she is suffering with a serious heart ailment, mere assertion that she has a good heart will not cure her.

Pain about the heart, going to the left neck, shoulder and arm, suggests angina pectoris. This patient had rheumatism fourteen years ago, which might have left a damaged heart. She has palpitation and dyspnea on walking and gas on the stomach, all symptoms of heart disease. The heart was under suspicion before, but in De-

cember, 1929, she suffered a sudden heart attack with pulse rate of 160, syncope, marked dyspnea, prostration, sleeplessness, cyanosis and pain about the heart. The doctor suspected coronary embolism. Could it have come from incipient crural phlebitis? Could there be coronary sclerosis? The attack began with pain in the leg.

When a heart with normal rate suddenly jumps to 160 and maintains that rate steadily, we are certain that it is due to paroxysmal tachycardia. Paroxysmal tachycardia sometimes occurs after acute coronary obstruction. Syphilis with plastic closure of a coronary artery, is also to be considered as a possibility.

We are dealing with a nervous, highly emotional patient. That does not exclude organic disease. A clot from the leg could not reach the aorta and coronaries, because it would not be able to pass through the lungs. I know of no case of cardiac infarct due to paroxysmal tachycardia. Can we have sclerosis of the coronaries in the early thirties? A man aged 39, who died of cardiac infarct, showed marked coronary sclerosis at postmortem. The normal electrocardiogram does not exclude acute coronary obstruction.

I think that this case shows the necessity of extreme caution in weighing evidence before drawing conclusions. While she has a history of rheumatism which might cause coronary disease, the attack was indefinite and the pain in the shoulder might have been due to neuritis. Her pain about the heart and in the left arm was constant from day to day and not sudden and of short duration, like true angina pectoris due to coronary disease. Palpitation and rapid heart are not symptoms of angina, as a rule. Generally the heart is slow and steady and the pressure high during the attack. Paroxysmal tachycardia does not initiate cardiac infarct. It may follow it.

This woman has two healthy babies and a negative Wassermann. That is against syphilis. X-ray shows an aorta of normal shape, width, and density. Generally with coronary sclerosis there is evidence of aortic sclerosis. Nearly all of her symptoms can be accounted for by emotional factors. The role of the emotions as a cause of heart symptoms will be discussed later.

When it was explained that the evidence for organic disease was entirely unconvicing, she began to improve. Some of her worst teeth were extracted and the rest given attention. A diet was prescribed for her gallbladder, and luminal for sleep, with quinidine to steady the heart. She was assured that, should her tachycardia return, we possess remedies to control and prevent it.

After spending the summer on the coast, she returned much improved, nervously composed, free from pain, and convinced that she has a good heart. The case shows how cardiac symptoms of a quite innocent nature, when misunderstood and misinterpreted, may start a fear complex with more and more heart symptoms.

Mrs. L. R., aged 50, was seen May 17, 1930. For the last four months she has had almost constant pain over the heart and sternum, which is made worse on stooping. There is some pain in the left arm. These symptoms are made worse on walking rapidly and she gets cold during the attacks. Sometimes the pain comes at night; for the last month it is worse on talking. Worry nearly always precedes the pain. The pain is not made worse by physical activity during the attack. She had similar trouble 20 years ago, after her first baby was born. At that time she went to the mountains and got worse. The pain has never been unbearable.

She is irritable, despondent at times, and cries a great deal. She recently inherited valuable property, which the government of Mexico is trying to take away from her. Her father died suddenly at 58, of angina pectoris.

Examination shows a woman of good musculature and youthful appearance, 5 feet 3 inches in height, weight 155 pounds. Eye and knee reflexes, active. No goiter. Blood pressure 110/84. Heart sounds all well produced and of normal intensity. No murmurs.

Fluoroscopic examination of the chest and the heart is negative for pathology. The electrocardiogram is normal in every way. Hemoglobin 85 per cent. Wassermann negative.

While the physical examination discloses no pathology, still the possibility of coronary sclerosis cannot be dismissed in a person of 50. The father died of angina at 58. She is emotional and gets cold during the attacks and they are not brought on by exercise, but worry may precipitate true angina.

Recently, S. A. Levine, of Harvard, tested with adrenalin thirteen cases of undoubted angina attacks. Pain came on within half an hour. The result of adrenalin in my case was entirely negative. So the evidence points to emotional or vaso-motor angina so far, but coronary sclerosis may supervene later. I think, on the whole, that the heart symptoms are of emotional origin due to her father's sudden death and her business and other worries. She was given a low cholesterol, low fat, low proteid diet in the hope of preventing coronary sclerosis, and the harmlessness of her present symptoms was fully explained.

C. F. B., an ex-service man, age 31, was first seen in May, 1925. He complained of pain about the heart, knife-life in character, constriction of the chest, choking sensation, and pain down the left arm. The attacks came on suddenly and the pain was very severe.

He was gassed in France in June, 1918, and was sent to the hospital, where a diagnosis of mitral stenosis and angina pectoris was made. He remained there until February 1919, when he was discharged with the same diagnosis, and was told by the ward surgeon that he would never be able to run again.

My examination showed no sign whatever of organic heart disease. His response to exercise was normal. All laboratory tests negative.

He was seen again Nov. 15, 1927: all physical signs negative. He was still having attacks every week or two and more severe; he was very nervous and sleepless.

A few days later he came to the office with pain in the heart and going down the left arm. He said that it had been almost continuous for four days. He was stooped over, holding and rubbing his arm, and seemed in great distress. I gave him 1/50 gr. of nitroglycerin sublingually, and in a few minutes the pulse fell from 120 to 96 and he got relief. He was last seen Oct. 9, 1929, again in the office. His pain had continued for a week. The pain had the usual distribution. The muscles of the left face were spastic, the left arm and hand cold and numb, the fingers white, and the hand cramped, with inability to relax it. Pinching the skin produced no pain. He said that the pain in his chest was deep seated and felt like the pulling of a hook in his heart. So far as the pain is concerned, the picture in many ways is that of angina due to coronary disease. There was relief by nitroglycerin.

Coldness and numbness and even anesthesia of the arm, may occur in true angina. The sudden onset is also characteristic. That he actually suffers cannot be questioned. I do not believe that he is malingering, though muscular cramps and anesthesia belong to hysteria at times. I believe that almost all of his symptoms are of emotional origin.

Against angina due to organic heart disease are: (1) No physical sign of heart disease. (2) His age: aortic insufficiency is about the only cause of angina under 32 years of age. (3) Cardiogram is normal. (4) The very prolonged attacks. Such prolonged pain might be due to cardiac infarct, but that could not happen so often. (5) Patients with true angina are seldom seen in the office. Walking, and even moving, makes it worse. Emotionalism and coronary sclerosis do not often go together. So I think this is a case of heart symptoms without heart disease. Ten years is a long time for such severe symptoms to continue if due to coronary sclerosis. At present, he looks much better and I have heard of no more attacks.

Mrs. X, age 34, was seen Aug. 19, 1930, complaining of shortness of breath, a smothering feeling, inability to sleep except when propped up straight, dizziness, and fainting spells. She says that at times she is just a nervous wreck, almost maniacal and feels like jumping in the river. Following worry, fright or overwork, she has spells with her heart which begin with a pain as though she had been hit in the epigastrium. This pain goes up over the heart and into the left shoulder and mid-scapular region and down the whole left arm. The pain seems to be inside the body. The first symptom is always lack of air and the chest feels as though it would burst. After the attack she often vomits and has crying spells. The hands and feet get icy cold and the lips and nails are blue. She is alarmed and fears death in an attack. Sometimes amyl nitrate, nitroglycerine, or morphine, gives relief; at other times they have no effect.

Her physician has told her that she has genuine angina pectoris, and to lie perfectly still, as even turning over might cause death. However, she has ridden five miles to see me when in an attack and has talked over the phone during other attacks, with no ill effect. She says that eating peppers or cabbage or other heavy food, gives relief. She talks excitedly and gives a very vivid description of her symptoms.

She has never been well since an attack of scarlatina at 14 years of age. She was delicate as a child and could not stand the summers in New Orleans. So she was sent to the Alps, but could not breathe at high altitudes. She was in bed for several weeks with inflammatory rheumatism when 7 years old. Some twelve or fourteen years ago she spat blood for a long time and was told that she had tuberculosis and could not live for more than six months. Our specialists in El Paso were unable to confirm this diagnosis. Some six years ago she had abdominal pain and other symptoms and was examined thoroughly, including an x-ray study of the digestive tract. She was told that she has gastric ulcer, gallstones, fixation of the appendix, and tuberculosis of the fallopian tubes, and that she would need several separate operations to cure her. It was finally discovered that she was three months pregnant and she got better without operation. During another illness she was told that she had a blocked ureter. On another occasion an abdominal exploratory operation was attempted, but she went "dead away" and no incision was made.

She does the housework and cooks for a husband and four children and is very active except when sick. Her father died of apoplexy. Her mother is living and well at 74.

She weighs 117 pounds and does not gain or lose. She has no goiter. Blood pressure is 120/80; heart rate, 80. The heart is normal in size and shape and there are no murmurs or other signs of heart disease. Fluoroscopic examination discloses no pathology in the heart or lungs. X-ray of the digestive tract shows gallbladder stasis rather marked but no stones. Electrocardiogram normal.

Discussion: We are struck at once by the fact that there is a great poverty of physical signs compared with all the serious ailments that have been discovered. How can we account for her symptoms? Has she true angina pectoris due to coronary disease? She has pain of that type and a very good doctor is convinced that such is her ailment. Her pain has yielded to nitroglycerin, and she has had scarlatina and inflammatory rheumatism, which may cause coronary disease. Syphilis with plastic closure of the coronaries could be present.

On the other hand she has had other ailments, apparently, of a very serious nature, which subsequently were not confirmed. At times morphine in repeated doses has failed to control her pain. I have never seen or

heard of it entirely failing in organic angina pectoris. Her attacks begin with dyspnea. Angina begins with pain and it generally is accompanied by rigidity of the chest and momentary inability to breathe. I have seen her in an attack and injected morphine which gave relief before it could have had effect, I believe, because her fears are dissipated. Is she a malingerer? Not at all, in my opinion. There is a real cause for all of her symptoms, but it is not to be found in organic disease. The emotional element in her case stands out above all else and is the starting point of her perplexing symptoms.

The emotions, such as anger, fear and rage, arouse the mechanism of self defense and lie deepseated in the primitive brain. Cannon and his associates have shown that the decerebrated cat will show fight and the expressions of anger and self defense, such as growling, showing of teeth, putting out the claws and bristling of the tail, immediately on coming out of anesthesia.

The emotional center lies in the hypothalamus, and here and in the thalamus all sensations are relayed before going to the cerebrum. The higher brain centers ordinarily exert an inhibitory effect upon the emotional center, but under great excitement this inhibition is overcome and the mechanism of self protection and defense comes into full play. The outward expression may be suppressed but the inward preparation for defense goes on. The sympathetic nervous system becomes highly excited, the pulse rate is increased, the blood reserve in the liver, spleen and splanchinic areas, is thrown into the circulation, the adrenals are stimulated, the arterioles are contracted, the blood pressure raised, the speed of the blood stream quickened, the sugar stored in the liver is mobilized, and the blood sugar greatly increased, while gastic secretion stops and peristalsis is suspended. Thus we see that the viscera, which are entirely beyond voluntary control, are reached and profoundly affected by the emotions. These facts enable us to trace the genesis of many obscure symptoms. We are here principally interested in visceral pain, especially in blood vessels.

From the time when Harvey touched and handled the ectopic heart of the young Count of Montgomery and found it insensitive so far as he could determine, up to modern times, the internal viscera have been thought to be insensitive. But more recent experiments demonstrate that they are sensitive to adequate stimulation. Most of the hollow viscera, and especially the blood vessels, are painful to stretching, overdistention and overcontraction. Puncture of a vein is painful, puncture of an artery still more so. Ligation of arteries is very painful at times. Spasm of blood vessels is painful, as seen in Reynard's disease and intermittent claudication. The pain of chronic arthritis, in some cases, has been found due to arterial spasm and has been relieved by sympathetic ganglionectomy, resulting in improved circulation and relief from pain.

The weight of evidence points to coronary spasm as the cause of angina pectoris. The Coffey operation of removing the superior sympathetic ganglion has cured angina pectoris. This ganglion has no sensory nerves. It is purely motor and, hence, the effect of its removal can be only one thing and that is to lessen contraction of the coronaries, the overcontraction of which is painful. Recent experiments of passing a ligature about the coronary artery in dogs, and pulling on it, give all the evidence of severe pain and also cause limping in the left front leg, indicating the same distribution of the pain as in angina pectoris in man.

Now to go back to the emotions and our cases. It makes no difference if a highly sensitized coronary artery becomes spastic because of an emotion or because of an overloaded stomach or physical exertion. The symptoms are much the same except for the emotional element or its absence. Even in true angina due to coronary sclerosis, the attack may be precipitated by emotional excitement. John Hunter, who had severe angina for twenty years, often remarked that his life was in the hands of any rogue who chose to anger or plague him.

That which has been said about the heart, applies to the other viscera, because the mechanism of pain in them is the same. Contraction, overdistention, pressure and pulling, cause pain and the result is very similar whether these conditions are brought about by local or organic disease or by overaction of the sympathetic nervous system under the drive of the emotions. It is the same mechanism that is brought into play, just as the fire department responds to a false alarm as promptly as to a great conflagration.

Our efforts in treatment must, therefore, be directed to tracing the source of the alarm and the removal of its cause. This is sometimes easy, as in the first cases recited in this paper. The frightened adult is no different than the frightened child, he must be shown that there is no danger. If the symptoms are of emotional origin they will disappear when all fear has been dispelled and the causes for emotional storms have been removed.

The first thing is a thorough examination that convinces the patient that no stone has been left unturned, and that no dire danger still lurks hidden somewhere. A good history, going into every detail, a complete physical examination, laboratory tests and especially x-ray studies and an electrocardiogram, help greatly in carrying conviction that no tumor, ulcer, aneurysm, or the like, has been overlooked. The genesis of the heart symptoms must be explained with absolute frankness and all questions answered honestly. Any evasion will lead to tha idea that, after all, the trouble is so bad that you dare not tell the truth.

Digitalis should not be given for two reasons: (1) because the heart is sound and it can have no good effect in any way; (2) giving digitalis increases the patient's conviction that he really has a bad heart.

Quinidine to regulate the cardiac rhythm does good because it helps to remove one cause of fear, the premature beats. But we must remember that, in cases such as have been disucssed in this paper, the whole trouble—the dyspnea; rapid, irritable heart; weakness and exhaustion; the cold hands and feet; and even the cardiac pain simulating angina pectoris—is due to the defensive mechanism brought into play by the sympathetic system and primarily aroused by the emotions. The moment that we get rid of fear the patient is cured.

CASE OF MASTOIDITIS, MENINGITIS AND BRAIN ABSCESS (CASE REPORT)

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(Present at the Staff Meeting of St. Joseph's Hospital, Phoenix, Nov. 10, 1930.)

Male, aged 21, admitted to St. Joseph's Hospital for the first time February 6, 1930, for operation on account of a subacute mastoiditis. A semi-radical mastoid operation was done at this time. As hearing was reduced to the minimal, and on account of certain pathological characteristics of chronicity disclosed previous to and at the time of operation, the patient was advised to have a radical. However, since he was very desirous of maintaining even a very small amount of hearing, a compromise procedure was agreed to and the above described operation was done.

The cortex was found to be very much thickened. The mastoid was mostly of the sclerotic type, with few pneumatic cells. Bone necrosis was encountered in both the superficial and deeper mastoid areas. The antrum was located high and deep in the mastoid structure. I refer to these pathological characteristics in order to clarify the subsequent course of this case. The above findings are prima facie evidence of chronicity and are very frequently the forerunners of grave sequelae. It might be added that the patient had an abscess in the right middle ear when a child of two years. He had a similar attack of otitis media in the same ear, for which I treated him, two years ago.

Following the mastoid operation, for a month after patient left hospital, the middle ear was dry and the postauricular wound was making a satisfactory healing. There reoccurred, however, some discharge from the middle ear. On April 26, 1930, he came to the office complaining of a very severe headache. The cerebral pains were not referred to any definite locality but were more or less generalized. The head pains became more and more aggravated and he was readmitted to St. Joseph's Hospital on April 28th.

Examination: Patient was extremely ill and restless and complained of severe pain in parietal and occipital regions. Pupils reacted to light and accommodaton. He was very much nauseated and vomited frequently. Ophthalmoscopic examination was negative at this time. General physical condition revealed no other abnormalities.

There was a small amount of non-pulsating discharge coming from a medium-sized perforation of right drumhead in its lower anterior quadrant. The operative wound showed some drainage following probing as far as the antral cavity. The culture taken from aural secretion showed the offending organisms to be streptococcus and staphylococcus. Temperature was 103.8. Pulse 90. Respiration 20. The temperature ranged, in the early part of his present illness, from 100 to 102.3 and pulse 80 to 90.

Laboratory examination: The urine showed a trace of albumin and a few pus cells. Spinal puncture was done immediately after admission. It was found to be under increased pressure, contained 760 cells per cubic centimeter and showed a hemolytic streptococcus. Spinal punctures were done about every other day as cerebral symptoms indicated.

On May 2nd, the fifth day after admission to hospital, the spinal fluid was found to be under greatly increased pressure and contained 1700 cells per cubic centimeter. The blood picture was not in keeping with the severity of the illness except that, on the 29th, his third day in the hospital, the leukocytes reached 19,400 with 87 per cent polys. On the second of May, the fifth day in the hospital, the leukocyte count was 14,800, with 86 per cent polys. Hemoglobin 80 per cent. Other counts recorded showed a lower leukocytosis but an increase in polys.

Dr. Kingsley, who acted as neurological consultant, concurred in the diagnosis of septic meningitis and it was thought wise first to combat the meningeal involvement, hoping thereby that a localization of a probable abscess might ensue. A blood transfusion was given the patient on the day of admission and another on the fifth day. The second donor had been immunized with an autogenous vaccine made from the spinal fluid of the patient. The vaccine contained hemolytic streptococcus. A polyvalent streptococcus serum was injected into the spinal canal after removal of fluid.

The condition of patient remained practically unchanged until May 7th, which was his tenth day in the hospital. At this time, examination showed that the right pupil was still larger than the left. However, both pupils reacted to light and accommodation. There was a slight amount of neck rigidity remaining. There were no pathological reflexes present and the Kernig's sign was much less marked. The temperature, until following the operation, ranged from normal to 100.8. Pulse dropped to 70.

Ophthalmoscopic examination: Right eye dis-

closed a grayish hue to optic nerve along the temporal border. There were no abnormal findings in the left eye. Patient complained of a diplopia. The diplopia was accounted for by paresis of the third nerve; and an involvement of the contracting fibers of this same nerve probably explains the persistent mydriasis. Patient steadily improved from this time, except for a very slight temperature. The discharge from middle ear remained about the same. On the 13th of May all reflexes were reestablished to normal. The general condition of patient was satisfactory. The spinal fluid cell count had dropped to 50 cells per cubic centimeter and there had been an absence of pathological organisms for several days.

The second operation was done on the 14th of May. After making skin opening, the previously operated cavity was found to be clean. However, on extending incision down to the middle ear and the aditus ad antrum, these cavities were found to contain considerable necrotic tissue. The ossicles were destroyed with the exception of the head of the malleus, which was bound down by the dense adhesions. Just posterior and internal to the inner border of the fallopian canal, was found a dehiscence in the roof of mastoid antrum, 3 by 2 millimeters in area. An exposed necrotic dura enmeshed in granulation was present. After probing of this area, a few drops of pus exuded. The radical mastoid operation was completed. No flaps were made and wound left wide open. Cigaret drain and iodoform gauze were inserted into operated cavity. From this time on, except for slight nausea and headache, patient made an uneventful recovery.

The bifocal vision due to third nerve involvement gradually subsided and the dilated pupil became normal in size. Another spinal puncture, done on the 16th day of May, three days following operation, showed the fluid to contain only 30 cells to a cubic centimeter. Another transfusion was done at this time, using an immunized donor. Before the patient left hospital I closed the operated wound with plastic flaps. The middle ear drainage gradually subsided and the ear was dry when epidermization was complete.

In commenting upon this case, there was manifestly a latent hemolytic streptococcus infection in the deeper recesses of mastoid. This was undoubtedly present for an indefinite period. This organism gradually eroded the bony covering of the temporal lobe, exposing dura and finally producing an intrameningeal abscess. When patient was admitted to the hospital, the abscess had advanced far enough to rupture into the cerebrospinal circulation, which was evidenced by the presence of a purulent meningitis. An immediate operation at this time might have resulted disastrously.

Spinal fluid drainage and blood transfusion were very important factors in minimizing the meningeal symptoms as well as localizing the infective focus. The use of blood from an immunized donor, or one recently recovering from a hemolytic streptococcus infection, is, I believe, a valuable adjunct in the treatment of this type of disease.

DISCUSSION

DR. SCHWARTZ: Statistics show about 80 per cent of brain abscesses are caused by infection from the middle ear and mastoid process. It is for this reason these cases are frequently seen by the otologist.

A common route of infection is by way of the thin plate of bone that separates the middle ear and mastoid antrum from the inside of the cranium. The infection may travel through the various channels, especially the diploic veins. The perivascular spaces are another frequent avenue of infection into the cerebral substance. Infective infarcts occur occasionally. They may or may not develop into a brain abscess. Some cases with obscure symptoms, that get well, can be explained by these infarcts. The abscess may be extradural or intradural.

Extradural abscesses are commonly located beneath the temporal bone over the roof of the antrum, or in the posterior fossa in the region of the sigmoid sinus.

Intradural abscesses have two sites of predilection in the temproal lobe and cerebellum. In Korner's series, 63 per cent occurred in the temporal lobe, and 31 per cent in the cerebellum.

The most characteristic symptoms are often entirely lacking. Before operation the symptoms may be masked by middle ear suppuration. The patient appears sick and has a mask-like facial expression. Headache is probably the most constant symptom. The pain is dependent upon the amount of intracranial pressure. As the intracranial pressure increases, the pain becomes more severe. Vomiting should arouse suspicion of an intracranial lesion., particularly abscess. The mentality is dull, and reasoning becomes slow and difficult. With increase of intracranial pressure, we shall have the characteristic symptoms of vomiting, pain, choked discs, and slow pulse. These symptoms are more common in brain tumor. Indentation of the visual fields is characteristic and the fields should be taken daily for any change. The field changes are due to the edema which accompanies the abscess. Nystagmus occurs in an involvement of the labyrinth or brain. Abscess in the functional areas gives rise to symptoms of definite localizing value. There are vast silent areas that may harbor extensive lesions without giving rise to any localizing symptoms.

When the abscess is suspected in the posterior fossa of midbrain, a lumbar puncture is not advisable. The patient might suddenly stop breathing. As a rule, the number of cells in the spinal fluid is increased. The treatment is entirely surgical. If a mastoid operation has been done, the pathological changes revealed may suggest what to do or where to go. It is well to become familiar with and follow the refined technic of the neurosurgeon. A timely operation and good technic may save a life.

The prognosis is always grave. The percentage of recovery is comparatively small.

In this particular case, Dr. McLoone is to be congratulated upon the excellent result obtained.

FRACTURE OF RIGHT FEMUR WITH SEPARATION OF THE LOWER EPIPHYSIS (CASE REPORT)

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(Discussed at the Staff Meeting of St. Joseph's Hospital, Phoenix, Nov. 10, 1930).

The patient, a schoolboy aged twelve, was brought into St. Joseph's Hospital on Sept. 8, 1929, with a fracture of the lower third of the right femur, a puncture wound of the right knee, which was draining synovial fluid. This condition followed an automobile accident which occurred on September 1st near Winslow, Arizona. The car in which

the boy was a passenger, and which was said to have been going about eighty miles per hour, turned over three times, throwing the occupants out, and this boy was thrown into a pool of water which covered him entirely. Had it not been for the immediate aid of passers-by he would have been drowned.

He was taken, with the rest of the passengers, to a doctor's office, was placed on the floor and later, when he revived somewhat, was placed in a chair. When the doctor came in he noticed the boy slumping down in the chair and his leg, instead of being in front of him, was around back of the chair legs. It was not until then that they were aware that the boy's leg was broken.

Physical Examination: Boy, aged twelve, lying on a board splint, which held the right hip, thigh and leg in good alignment. There was marked swelling of the right thigh and knee, with marked discoloration and ecchymosis and puncture wound of the right knee, from which was exuding a fluid resembling synovial fluid. The general examination was essentially negative except that the pulse was rather rapid and the boy looked tired from his trip. There was a pustular eruption on his face and hands—probably an impetigo.

The patient was placed in a Thomas Splint with a Pearson attachment and the thigh and leg suspended, with the knee in a flexed position.

X-ray, Sept. 10, 1929, showed wide separation of the fragments of the femur, and the proximal fragment internal and anterior to the distal one. There was also separation in the epiphyseal line of the femur, with some displacement of the fragment.

Sept. 11, Dr. Palmer saw the case in consultation and it was decided to do an open reduction, as it did not seem possible to reduce the femur by the closed method as this had been diligently tried in Winslow. Because of the condition of the patient, it was decided to wait a few days for him to recuperate somewhat.

Sept. 16, open reduction was done. There was a large amount of clotted blood found about the ends of the fragments with considerable laceration of the soft parts and the muscle tissue between the fragments, which prevented the reduction by the closed method. The fracture being only slightly oblique and with rough ends, it was decided to place the fragments end on and not put in any internal fixation, because of the hematoma and the marked contusion of soft tissues. The case was put in a cast from toes to rib margins and to the knee of the left side.

Oct. 8, x-ray showed fragments in apposition but with external bowing at the point of fracture. There was definite hard callus along the inner side of the fracture.

Oct. 9, patient was taken out of the cast and put up in traction abduction.

Oct. 10, under gas anesthesia, the bowing of the femur was straightened out and the thigh and leg left in traction abduction. It was thought that it would be possible to hold the fragments inward with the application of Pearson pads. This method was tried for some time but without very much success. The wound opened and a superficial infection developed, probably from the skin infection that the patient had. The continual adjustment of the pads kept continued movement in the fragments and the union did not become firm.

October 29, there is another x-ray report and a "dirty dig" from a radiologist who signs himself "W.W.W.": "We have tried to secure the desired information on this patient by means of a lateral projection taken with stereoscopy. The only thing that we are able to determine is that the alignment is good. Bone detail is obscured by splint and we are unable to get at the patient in a position which would permit anteroposterior view. Some other arrangements will need to be made before we can secure roentgenograms of this patient in the location, and with the scaffolding which he has around him at the present time."

We decided to treat the case as best we could until the infection cleared enough to be safe to put in a Steinman nail and, by means of skeletal traction, to get a better alignment. We could not get traction enough, even with the knee flexed, to overcome the bowing bcause of the separation of the lower epiphysis.

Nov. 30, x-ray roentgenograms of the right femur, taken with portable machine, show the fracture in the middle portion of the bone with considerable outward angulation; fragments of the bone apparently in good position, and there is evidence of bony callus being deposited, although the point of fracture is overlapped with the rubber pressure pads. There is also fracture at the epiphyseal line which shows some increase in density of the distal end of the shaft, probably representing attempt at healing.

Here, again, we did not get the information we wanted, the rubber pads being in the way. However, there was not union as there was mobility at the site of fracture.

Dec. 9, 1929. Although the infection was not entirely cleared up we decided not to wait longer, and put a Steinman nail in the lower end of the femur above the epiphyseal line, put on fifteen pounds weight, and in a short time began to move the knee.

Dec. 30, 1929: Roentgenograms of this patient's thigh and knee, taken with the mobile x-ray unit, show good position and alignment, with extensive callus formation along the internal and posterior aspect of the bone, extending well above and well below the fracture of the femur. There is evidence of union at the piphyseal line.

Jan. 18, 1930, the Steinman nail was removed.

Jan. 26, the leg was taken out of the splint.

Jan. 29, the patient was up in a wheel chair.

Feb. 24, 1930, patient was discharged. Union firm, slight angulation at the knee. April 1, patient was bearing some weight but the knee could not be extended to the normal potition. May 1, patient was walking without crutches, knee almost straight.

Aug. 1, 1930, patient walking without limp but slight knock knee.

CASE REPORT.

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(Presented at the Staff Meeting of St. Joseph's Hospital, Phoenix, Dec. 8, 1930).

Male infant, born in St. Joseph's Hospital, September 18th, and died October 8th. Family history negative; father and mother both well. Wassermann on mother's blood was negative. No brothers nor sisters. One brother died last year at age of three days. (Death was due to enlarged thymus. X-ray showed- large thymus. One x-ray treatment was given. Baby became cyanosed and died in cyanotic attack.) Record states that this second baby was a normal delivery but that the baby was covered with pustules at birth. These lesions seemed to improve. The baby nursed well and by the sixth day had re-

gained its birth weight. On the sixth day it was noticed that the left breast was swollen and red. This was opened by the physician and a bloody purulent fluid escaped. Culture of this fluid showed staphylococcus aureus. The temperature ranged from 99 to 100.8 degrees. At this time, the skin eruption on the body had cleared up except upon the buttocks. The drainage in the breast gradually became less and had ceased, but the breast was still swollen and red. The baby left the hospital on the 13th day, apparently in fairly good condition, although it was still 3½ ounces under its birth weight. The baby seemed to be doing well at home until the third day, when it commenced to cry, especially when the head was moved. It cried as if in severe pain. There was some edema and tenderness over the occipital region. This swelling rapidly involved the entire scalp, and the baby was taken back to the hospital. Consultation advised reopening of abscess in the breast; also, that erysipelas antitoxin be given and free opening of any other abscess which should occur. There was a questionable fluctuation in the occipital temporal region. Three doses of erysipelas antitoxin were given at twelve-hour intervals, with no apparent improvement. The following day, there was a distinct fluctuation in the left occipital temporal region. This was freely opened and a large amount of thin bloody sero-purulent secretion escaped. This was cultured, as well as the purulent fluid from the breast. Both showed staphylococcus. A hard indurated cellulitis developed on both sides of the neck, extending around in front, completely surrounding the neck girdle. There were no definite fluctuating areas. One area seemed a little softer and was incised, but only a small amount of serous fluid escaped. The baby commenced to have clonic convulsive seizures. Spinal puncture showed a clear fluid with some increase in pressure. There were four cells and no organisms were found. These convulsions became more frequent and the babe died on the fifth day of his second admittance to the hospital. An attempt was made immediately after death to obtain a specimen of heart blood for culture. This was unsuccessful but a clear paricardial fluid was obtained, which contained no organisms.

The autopsy report was as follows: The body is that of a male babe showing evidence of a large abscess involving the left breast, which has been opened by incision, and another abscess under the left ear, which has been incised, and a large area of induration below the right ear extending over the right side of the neck. There is a thickening and induration of the scalp. The left lung was removed. There was a small amount of pleural fluid present. The lower lobe showed passive hyperemic—right lung very dark in color—lower lobe increased in volume and of firm consistency, evidently a pneumonic consolidation. There is one large dark area with a gray striation just beneath the pleura. The thymus shows moderate enlargement, with slight excess of pericardial fluid. Heart is normal in size. The valve leaflets, both tricuspid and mitral, show reddening and a slight irregular roughened appearance, mostly representing an early endocarditis. The liver and spleen were normal. Left kidney pelvis is enlarged with narrowing of the cortex of the kidney, representing a slight degree of hydronephrosis. Sections of lower lobe of right lung show complete consolidation. The air vessels filled with blood cells and inflammatory exudate: pus cells and edema with multiple miliary lesions of necrosis. Septic pneumonia. Examination of the left ureter shows almost complete obstruction of the lumen at its junction with the bladder. Apparently congenital narrowing and is the cause of the hydronephrosis of the kidney.

Anatomical Diagnosis: Septic pneumonia; erysipelas of scalp; cellulitis of both sides of neck; abscess, left breast; possible early endocarditis.

The septic pneumonia which was found at the autopsy might have been an insufflation process, as the baby expectorated similar bloody purulent material. It is possible that the marked cellulitis of the neck might have been discharging somewhere in the throat. New-born infants bear infections very poorly. For that reason, our nurseries are extremely careful to protect the babies from colds, skin infections and other contagious diseases, for, not uncommonly these terminate fatally. It would seem that this was a blood-borne infection. Just where the staphylococcus first gained entrance is problematical. The septic pneumonia, beginning endocarditis, widely separated absceses, would warrant a diagnosis of septicemia, without a positive blood culture.

ENURESIS

Review of Current Literature

O. H. BROWN, Phoenix, Ariz.

(Continued from December Issue)

J. Jacobavici, C. I. Urechia and E. Tepasu[36] report 19 cases of urinary incontinence and nocturnal enuresis, in which the finding was made in 16 of occult spina bifida. Operation was done on this group with good results. They say education should first be attempted especially in the mentally deficient, and where this fails a large per cent should be operated upon.

G. J. Mohr and E. H. Waterhouse[36] found the causes of enuresis in 15 children to be poor training, emotional factors, physical factors, suggestion and fatigue. They conclude that a neurosis is frequently a fundamental condition. The treatment must be based upon the etilogic factors and must be highly individualistic.

Wm. S. Ezickson[37] had two serious cases of enuresis, one in a boy six, with a duration fo two years and the other a boy eight and a half with enuresis since babyhood. The first case was found to have a calon bacillus infection of the urine with a large amount of pus. A vaccine was prepared and administered; the child's urine gradually cleared of pus and after about two months there was no more enuresis; the other case was found to have occult spina bifida. Treatment of this was not permitted.

Lemoine[38] had one case of urinary incontinence associated with fecal incontinence as a result of meningeal cyst of the lumbar region. Diagnosis was made by iodized oil and x-ray. Removal effected cure.

Arthur I. Blau[39] studied 90 cases of enuresis. Thirty-eight cases were of ages four to six, 28 were over 10 years. Forty-one were male and 49 female. Two of the cases were diurnal in type, 69 were nocturnal and 19 were both diurnal and nocturnal. There were a variety of findings such as enlarged tonsils, adenoids, anemia, caries, malnutrition, phimosis and bronchitis, but correction of the abnormalities were without great benefit. The duration of enuresis before treatment was instituted, was less than one year in 10 cases, less than two in 28 cases, and over two years in 37 cases.

W. Howell[40] says the bladders of children are sen-

sitive to reflex stimuli, local irritation and sensory stimuli of various sorts, various psychic acts are prone to cause a contraction of the bladder.

A. Friedell[41] has had three cases of enuresis in which there was reversal of the day and night specific gravity of the urine, and hence with undue nocturia.

S. A. Moss[42] says enuresis may result from reflexes caused by local irritation from phimosis, adherent foreskin of clitoris, balanitis, epispadis, hypospadis, vaginitis, renal or vesical calculi, pyelitis, cystitis, tuberculosis of the genital tract, pinworms, lack of cleanliness, skin diseases of external genitals, etc. He also says masturbation, spina bifida, myelitis and nocturnal epilepsy may be causative factors.

Pfister[43] says that the type of nocturnal enuresis which is the result of epilepsy occurs relatively infrequently.

E. Pototsky[44] thinks enuresis occurs especially in the psychopathic children and divides them into four groups, the obstinate children whose lack of inhibition is due to desire to annoy the parents, the highly anxious children who have undue fear of punishment, the children with decreased capacity for attention and concentration and children excessively indifferent to all forms of impressions.

D. Patterson[45] found that 75 of his cases had undue frequency of urination and hence tended to evacuate the bladder at the slightest provocation.

Pierre Delbet and Andre Leri[46] have operated upon occult spina bifida in several cases of enuresis, and effected prompt cures.

F. O. Pybus[47] says enuresis has been a common symptom in cases of spina bifida which he has seen.

J. S. Leopold[48] had a girl age six with spina bifida and enuresis in which operation of the spina bifida cured the anuresis.

C. Beck[49], Von Recklinghausen[50], Mr. Barst[51], F. Hesse[52], and J. Van Finck[53] have written extensively upon spina bifida and enuresis.

Maurice Ostheimer and I. V. Levi[54] claimed that 80 per cent of their cases of enuresis were benefited by the use of atropin used to the point of toleration.

Knotz[55] recommends a vegetable diet for enuritic children because by it a large amount of the water of the body is carried off by the bowel; he restricts the fluids after 4 p. m. and raises the foot of the bed and endeavors to make the child sleep upon his side, tying a brush to the child's back if necessary, to make him uncomfortable if he gets onto his back. He says most of enuretics are excessively heavy sleepers and need to be waked up at intervals during the night. He recommends a two-hour sleep for morning and for afternoon.

Lewis[56] advises putting the patients on a strict anti-diabetic diet; his results have often been excellent from this regime. He also uses tonics.

A. Collin[57] finds enuresis is sometimes a result of digestive disorders often from over eating. He advises mild laxatives, restriction of diet, hot compresses and light massage to abdomen.

Klotz[58] prescribes cold douches to the anal and perineal region in a specially built chair.

Cipriati[59] recommends the diathermic current.

Baruch[60], Watson[61], and others testify to the value of belladonna.

Fleischner[62] uses belladonna in suppositories.

J. Allan[63] says there are two varieties of enuresis, the atonic and that characterized by vesical irritability. In the vesical irritability type belladonna is highly useful, and should be used until there is dryness of the mouth, mydriasis, disturbances of vision, etc.; for a child five years of age 10 minims of tincture of belladonna may be given three times daily and increased two minims every few days, until tolerance is determined.

Simpson[64] has used tincture of belladonna three times daily in doses as large as 20 to 25 minims. He advises neutralization of hyperacid or strongly alkaline urines, thinks strychnine may be a serviceable tonic and has used belladonna; if infection is found in the urine it should be eliminated.

Herman[65] had his patients urinate at regular stated intervals, interrupting the streams every little while until the bladders were empty. This was done for the purpose of developing a conscious control of the bladder evacuations. It should be done at first under supervision of the physician.

Haven[66] cured two 18 year old girls by gradually distending every other day their bladders with four per cent boric acid solutions. The bladders in both cases were abnormally small.

Sir Henry Thompson[67] instils silver nitrate solution into the posterior urethra and claims it is beneficial if used in a strength to produce smarting.

J. Wilson White and Edward Morton[68] say that nocturnal enuresis of the type due to carelessness or laziness can often be successfully treated by awakening the children each night an hour or so in advance of the time they are likely to void in bed.

Wachenheim[69] treats enuresis as a "tic convulsif;" i. e. the child must urinate at regular intervals during both day and night. When this does not cure he cuts off all late afternoon and evening fluids and reduces the evening meal. He thinks atropin may be beneficial as may tonics, removal of adenoids and tonsils, etc.

Freyberger[70] advises a regulation of the diet, sleeping on a hard mattress, light coverings, cold sponging along the spine and taking the children up once or twice during the night to pass urine.

Valentine and Townsend[71] used Fernand Cathelin's method of injecting 10 c.c. of saline solution into the "sacral canal between the periosteum of the vertebrae and the dura mater." The patient is placed in Sims' position and by using the last palpable spinous processes of the sacrum the needle is introduced into the sacral canal.

Jabouilay[72] obtained favorable results in several cases by retrocecal injections of sterile saline injections. Several injections were often necessary.

Allaria[73] applied Cathelin's method of epidural saline solution injections into the sacral canal in 22 cases and obtained good results in about half the cases, all he thinks entirely from suggestion.

Cahier[74] injected 60 to 70 c.c. saline solution subcutaneously into the anterior perineum to make a swelling the size of an egg with slight pain and obtained satisfactory results.

G. Sisto[75] had excellent result in a child 14 years old, afflicted with enuresis all of his life, by Cathelin's epidural injection of saline solution.

Zanoni[76] treated 134 cases of enuresis with suprarenal gland substance and cured 66 cases, improved 21; 14 were better during the period of treatment; 21 were unchanged. He thinks the treatment was not continued a sufficient length of time in his failures.

Hertoghe[77] says nocturnal incontinence is due to thyroid insufficiency and a per cent of his cases were benefited by administration of thyroid extract. He also gave potassium iodide and bromide.

Leonard Williams[78] cured 25 cases by giving them small doses of thyroid extract. He says it is extremely important not to give large doses as such may cause enuresis.

Fleischner[79] treated ten patients with thyroid ex-

tract, cutting out fluids after four p. m. and elevating the foot of the bed. Eight were improved and in four of them circumcision had previously been done without benefit.

McCready[80] recommends supervision of the child's exercise, diet, and bathing, and guarding against physical or mental strain. He also gives thyroid extract.

A. C. D. Firth[81] treated 28 cases with thyroid extract and obtained cures or marked improvement in 16.

John Rehrah[82] reports that of 716 cases of adenoids operated by Fisher, 14.9 per cent had enuresis; that Mygino found enuresis in 7.75 per cent in 500 cases and Gruback 14.28 per cent in 427 cases; he also says Hamonic circumcised 187 cases of incontinence with cure of 130. He has used thyroid extract, fluid extract of ergot and atropin with good results.

Samway [83] claims good results from painting collodion over the outlet of the urethra. The heightened intraurethral pressure awakened the child.

Gersung[84] injected paraffin along a relaxed urethra, especially for atonic sphincters.

Semb[85] reports that the outcome in one of the cases treated by Gersung by paraffin injections along the urethra and in another treated by him was good years afterwards. In one case the urethra had been lacerated by childbirth and repeated operations had failed to effect a cure.

Fabre and Trillat[86] injected solid paraffin for severe incontinence due to trauma of the urethra, with perfect results.

E. H. Eising[87] treated two cases of intractable incontinence with paraffin injections along the urethra[88]—one injection in one case and three in the other—with cures.

Robt. K. Rewald[88] reports that his cases of enuresis were otherwise healthy, well-nourished and intelligent and but few were neurotic. He stresses another point, which many authors mention, that the physician should have the full cooperation of the child and his parents in treating enuresis. He uses general measures, drugs and suggestion in treatment. His general measures are, limited fluid after 4 p. m., evacuation of bladder just before retiring, getting the child up and awake at intervals through the night, and compelling him to walk to the bath room. In drugs he has no confidence, except in atropin. This he uses in a 1-1000 solution in ascending doses to the point of tolerance; for the diurnal and nocturnal type he gives doses at 10 a. m. and three and seven p. m. For the nocturnal type doses at 10 a. m. and three and seven p. m. The first dose is one drop and this is increased daily until short of the point of tolerance. The maximum dose is maintained for a period of from two to six weeks and these gradually reduced. He admits that psychiatrists scoff at the value of atropin but he is thoroughly convinced that it is of much help; in cerain cases which were doing well under the atropin he substituted water and the cases promptly relapsed; they improved again as soon as the atropin was resumed. He repeats the course of treatment whenever necessary. One case was treated ten times in four years before a permanent cure was effected. He approves of suggestive therapeutics.

Bronson Crothers[89] tells the enuretic that he can stop his bed wetting if he wishes to and each specific case is told that he must wash the bed clothes each morning after they had been soiled. He has the mother promise the child 25 cents for each night in which he remains dry for the first month and a dollar a week thereafter for a time. One child fell from grace twice the first week and once the second and has been dry ever since.

L. Coffe, Bainglas and Comsa[90] report the use intravavenously of methenamine in a group of cases with involuntary micturition from a variety of causes, with satisfactory results in a small per cent of the cases.

Francis B. Jacobs[91] uses pituitrin as a routine measure in enuresis. He treated 50 cases; 17 were cured; 17 were geratly benefited; 16 were not benefited.

Harry Lowenburg[92] admonishes physicians against promising their patients that removal of tonsils, adenoids, and correcting other abnormalities by surgery will cure enuresis.

A. R. Thompson[93] recommended stretching the bladder with a 75 c.c. column of salt solution but the method has not stood the test of time.

C. Bruns[94], F. Korats[95], Gallaria[96], and P. Mueller[97] have demonstrated to their satisfaction that psychotherapeutic measures are fully as serviceable whether used without or with drugs or placebos.

Editor[98] recommends for the treatment of a man, age 25, who had suffered from bedwetting for six years, x-ray examination to discover if spina bifida exists and epidural injections of an indifferent fluid as sodium chloride to relieve enuresis.

H. Cramer[99] reports on Goebell's operation for enuresis, and says his results have been good. The upper part of the neck of the bladder are grasped from behind and pulled upward and forward by a loop which is fixed to the fascia of the anterior abdominal wall. The operation seems to be for those cases of enuresis due to local abnormality—usually lacerations.

G. Brown Miller[100] advises operation upon the urethra where incontinence is due to lacerations of the urethra, provided there is adequate tissue to offer hope of cure; where operation is futile paraffin injctions may give good results.

Caspar[101] advised cauterization in two or three deep grooves of the vesical sphincter through a vesical cystoscope with reasonable hope of cure.

H. A. Kelly[102] describes in detail an operation for repair of a lacerated bladder sphincter.

E. L. Young[103] gives the operative procedure for treatment of incontinence when associated with or dependent upon epispadial and other conditions.

Cumston[104] thinks there is virtue in the usual medical treatment of enuresis and especially recommends the instillation of a silver nitrate solution into the urethras.

W. E. Carter[105] approves of the recommendation of Sachs that treatment for enuresis is largely a matter of habit training.

A. Mattes[106] says from his studies the causes may be grouped under three heads: Psychic, neurogenic and mechanical. He says there may be a combination of any two or all three of these factors. For treatment he recommends, alkalinization of urine, reduction of fluid intake after 3 p. m., last meal never later than 5 p. m., no sweets nor pastries in afternoon, and voiding before retiring. If improvement is not prompt he prescribes sodium bromide and tincture hyoscyamus. If needed, he uses silver nitrate instillations, (two to six per cent), every second to fifth day, for about ten times to increase the tone of the vesical sphincter. The urethra may be advantageously dilated. He reports a series of 20 cases successfully treated.

J. R. Stamper[107] especially recommends early treatment, the use of thyroid in hypothyroid cases and the correction of whatever local pathology may be found.

H. W. E. Walther[108] advises the use of infant cys-

toscopes in order to properly make the diagnosis. A catherterized specimen of urine, specially in little girls, should always be studied. He advises the use of one holf to two per cent silver nitrate—one to two drams—instillations into the bladder.

Frank J. Chalaron[109] says he has seen obstinate cases of enuresis in soldiers or would-be soldiers in which all ordinary treatment had failed. He used treatment first recommended by Cathelin, consisting of epidural injection of 10 c.c. of a sodium chloride solution. In three men this method produced marvelously successful results. A young girl had to have two epidural injections to effect a cure.

Porter[110] uses a method suggested by F. L. Dunham which consists of a card on which are printed four lines as follows: "I will not wet the bed. I will wake up at 10 o'clock. I will get up and pass water. I will not wet the bed any more." These four lines are to be repeated ten times during a diurnal urination and again ten times just before going to bed. The child should be awakened at 10 o'clock if he does not awake of his own accord, and made to walk to the toilet and urinate. The mother has a blue card and red and gold stars. For each night successfully passed the child is allowed to paste a red star on the card and for each week a gold star. Allowing the child to report his success by telephone to his physician may be beneficial.

C. Wile[111] recommends the formation of classes of enuretics in dispensary practice so that all may be trained simultaneously.

Dunham[112] emphasizes the importance of having the child repeat sentences to the effect that he will not micturate in bed, etc.

Arthur I. Blau[113] says the drugs used in a series of cases were strychnine, ergot, atropin, belladonna, and many others, but the results were unsatisfactory. Pituitary extract was used both by mouth, and hypodermic. About 75 per cent of the cases treated with pituitary substance were cured.

Bonacarse[114] reports eight of 12 cases of enuresis treated with pituitary extract were cured and three others were greatly benfited. Atropin and massage of the bladder were the only measures that Adrien Bleye[115] found effective in the treatment of true enuresis. In the main however, bladder massage was effective only against diurnal enuresis.

I report from among a number that I have treated two cases of nocturnal enuresis in boys about 16 years of age; one was successfully treated by the administration of potassium bromide. There seemed to be no abnormality in this case except that the bladder muscles responded too readily to a full bladder. My idea in this treatment was that the bromide would lower the sensibility of the cord centers which respond to a full bladder by evacuating it so that the bladder might not empty itself before the child awakes in the morning.

The other case seemed to be one of a diabetes insipidus type. He was given pituitary extract and after several months' treatment obtained a cure.

The case that prompted this study of the literature seems to be a case with hypersensitive, untrained spinal cord, centers. He has not been improved by bromides and restriction of fluids of late afternoon and evenings. He has tried putting an alarm clock timed to go off to awaken him before emptying of bladder would occur but he sleeps through without hearing the clock. I have had him reduce his school work and take a two hour sleep each afternoon with the plan that he is to study till 10 each evening. He is to empty his bladder before going to bed. His metabolism test is normal and I have discovered no local abnormalities. I shall x-ray his back for occult spina bifida, and shall have him use mental therapy. I shall try atropin if the suggested methods fail.

It has seemed to me that protein sensitization reactions of the bladder might cause a hyperirritable bladder and hence be largely contributary to enuresis in certain cases.

SUMMARY

Enuresis is probably most commonly the result of inadequate training in bladder control. It would seem that the conscious centers have been at fault in not exerting sufficient inhibitory power to prevent evacuation, or more especially to overcome the thought of the desire to evacuate. There is little doubt but that certain bladders are never stretched to the same extent as are others. Heavy sleeping and fatigue seem to be responsible for nocturnal enuresis in a proportion of cases; laziness and not-caring may be factors in occasional cases. The lower cord centers may be hyper-irritable because of such abnormalities as occult spina bifida, tumors, etc.

The bladder and posterior urethra may be hypersensitive and the bladder muscle hypertonic, so that the desire to micturate is caused by even a small amount of urine in the bladder. The urethra and sphincter may be abnormal so that there is little or no possibility of normal control of the outlet of the bladder. There may be local infections and irritations of the genital tract rendering the bladder hyper-irritable. There may be abnormalities in the body elsewhere than in the genito-urinary system which may reflexly affect the bladder as its nervous control.

The treatment seems to be predominantly habit training and mainly by mental therapy. The patient must be told that he can stop his bed wetting if he tries hard enough, that success will come after a period of trying; he must determine that he will not sleep heavy and he must do everything possible to prevent excessive fatigue; he must determine that he will awaken when a desire to micturate becomes strong. If he will but analyze the problem thoroughly and use his will power to overcome it he will usually succeed. All and every method of treatment has an element of mental therapy in it. The enuritic child should not be allowed to become too much fatigued by day; morning and afternoon sleep periods may overcome too heavy sleeping at night. All inflammation and irritation in and about the genital tract should be given careful treatment. The use of bromides to dull the cord centers seems entirely logical. The administration of atropin to relax the involuntary muscle of the bladder also seems rational. I also can see no sensible objection to restriction of fluids in late afternoons and evening.

Pituitary, thyroid, suprarenal or other internal secretion extracts in cases where there are signs of hyperactivity of the respective glands seem to be logical therapy when used especially with the preceding suggestions. Pituitary extract particularly where a large amount of urine is being evacuated it seems should have curative possibilities. Likewise in appropriate cases an antidiabetic diet would seem rational. In cases where protein sensitization of the bladder or cord centers exists a careful regulation of the diet would certainly be indicated. It is always well I am sure, that enurities should not eat excessively. Teaching the enuritic to control the premature desires to micturate and to stretch the bladder would seem to be a great benefit in certain cases. Operative procedure for an occult spina bifida should be undertaken when such condition is proven and all other methods have

failed to cure. Likewise operations on lacerated and traumatized sphincters are indicated when there is no hope from other procedures. In event of failure from operation there may be hope through paraffin injections along the urethra. Abnormalities such as tonsils, adenoids, and other diseased areas should be corrected but the procedure should not be held out as a cure for incontinence. The painting of collodion over the urethra outlet seems well worth trials. A string should not be placed about the penis is in one instance of which I read gangrene resulted. The instillation of silver nitrate into the bladder to the point of causing smarting on micturition might be worthy of trial as I am sure it might sometimes be counted upon to beat an alarm clock for awakening a person. I can see little if any excuse for epidural or perineal or retrocecal injections of saline. Last but not least parents should be educated to the understanding that children can be taught early in life to get control of bladder and intestinal evacuations. The early weeks of the child's life is none too early to begin the training process. The proverbial bedside pot has its place and should be used constantly. The child can early be made to feel that he has disgraced himself when he has soiled himself. The fault for enuresis is usually more in the parents than in the children.

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St. Joseph's Hospital (Phoenix)

(November Staff Meeting)

St. Joseph's Hospital Staff met in joint meeting with the Maricopa County Medical Society on Monday evening, November 10, 1930, at 8 p. m. The County Society presented their program and at 9 p. m. the meeting was turned over to the staff, Dr. Brockway presiding, with thirty-eight members present.

The meeting was called to order by Dr. Brockway and the minutes of the previous staff meeting and the previous executive council meeting, also the analysis of hospital service, were read by the secretary.

The purpose of recommendation of the council, that the office of emergency surgeon be done away with, was explained to the staff by Dr. Brockway.

The following applicants were voted to membership by the staff.

Loren C. Barlow, M.D.; Martin C. Flohr, M.D.; Robert Franklin, M.D.; Guy French, M.D.; V. F. Kennedy, M.D.; Hilary Ketcherside, M.D.; Leslie R. Kober, M.D.; J. D. Hamer, M.D.; M. I. Leff, M. D.; William Maloney, M.D.; John H. Patterson, M. D.; Norman Ross, M.D.; Oscar W. Theony, M.D.; W. A. Baker, D.D.S.

The scientific part of the program was then presented, the program being prepared by Dr. W. A. Schwartz.

1. Case No. 18979. Fracture of right femur with separation of the lower epiphysis, presented by Dr. Greer, with general discussion.

2. Case No. 18827. Brain abscess and meningitis, presented by Dr. McLoone, discussed by Dr. Schwartz.

Southwestern Medicine

Printed by THE A. C. TAYLOR PRINTING CO., Phoenix, Arizona
Published monthly for the Board of Managers of the four constituent societies.

Volume XV | JANUARY, 1931 | No. 1

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COMPLETION OF THE GRUNOW MEMORIAL BUILDING IN PHOENIX

The completion of the Grunow Memorial building in Phoenix the latter part of December has allowed the physicians and surgeons who will have their offices there, and who will form the Lois Grunow Memorial Clinic, to move in. The building of this institution and formation of the clinic group was the culmination of plans laid by Mr. Wm. C. Grunow of Chicago and Dr. W. O. Sweek of Phoenix, about a year ago. Because of the dominant influence of Dr. Sweek in the formation of this organization, it is more familiarly known in Phoenix and vicinity as the "Sweek-Grunow Clinic." The plans include research laboratories to be supported by rental from the offices and profits from the commercial laboratories connected with the clinic group. Mr. Grunow's gift embraces the building and equipment, including the laboratory equipment. The doctors in Phoenix who will have offices in the building and participate in the work which is performed as a clinic organization, are the following:

Dr. W. O. Sweek, organizer of the project, who will have charge of all the surgical work; assisting him will be Dr. Guy C. French, who has been associated with Dr. Sweek for the past two years; they have moved from the Heard Building to the new structure.

Dr. Win Wylie, who has moved from his offices in the Goodrich Building, will be consultant in diseases of women for the clinic group.

Dr. Floyd B. Sharp will continue in the practice of the specialty of obstetrics, having moved his offices from the Heard building; associated with him will be Dr. Martin C. Flohr, who has recently completed his residency work at St. Joseph's Hospital.

Dr. William A. Kingsley, formerly located in the Heard Building, has moved to the new structure, and will continue in his specialty of eye, ear, nose and throat disease.

Dr. W. P. Sherrill, formerly in the Luhrs Building, has moved and will continue in the practice of pediatrics.

Dr. H. J. McKeown has moved from the Heard Building and will continue to practice the specialty of chest diseases.

Dr. John A. Lentz has moved from his down-town office and will practice dentistry from the new location.

The physicians, not previously practicing in Phoenix, who will have offices in the building and form part of the clinic organization, are the following:

Dr. L. B. Baldwin, from Rochester, N.Y., who will practice in the field of internal medicine.

Dr. David M. Davis, from the Brady Urological Institute, Baltimore, will practice urology.

Dr. Percy Brown, formerly of Harvard University, will be the radiologist for the group.

Dr. Paul H. Guttman, formerly of Minneapolis, Minn., will be in charge of the clinical laboratory.

The doctors in the new building will all conduct their private practices as heretofore, functioning as a clinic organization on those cases which come to the institution for that type of service.

ST. JOSEPH'S HOSPITAL (Phoenix)

(December Staff Meeting).

The St. Joseph's Hospital Staff and the Maricopa County Medical Society met December 8, 1930, in the Lecture Hall of the Nurses' Home, with fifty-three doctors present.

The program of the Maricopa County Medical Society was presented first, consisting of a talk by Dr. William H. Walsh of Chicago.

The staff meeting was then called to order by Dr. Brockway and the minutes of the November staff meeting were read by the secretary. The proposed Constitution and By-Laws were then discussed and Dr. Brockway, chief of staff, proceeded with the reading of the proposed Constitution and By-Laws to and including the third article. There was some discussion upon Article 3, relative to membership.

There seemed to be some confusion in the minds of the members present and Dr. Holmes moved that each staff member be furnished with a copy of the proposed Constitution and By-Laws and that the matter be taken up at the next regular meeting. The motion was seconded by Dr. Shields and was carried. Later, Dr. Victor Randolph moved that this motion be reconsidered and that the members be furnished copies immediately, that the staff meeting be adjourned to Monday night, December 15, and that the discussion of the Constitution and By-Laws be continued at that time. This was seconded by Dr. McLoone and the motion carried.

The scientific part of the program was then proceeded with and Dr. Jordan's case, No. 20824, was presented. Discussion was offered by Dr. Carson and Dr. Palmer. Dr. Carson especially requested a discussion of his discussion for the reason that some recommendations for the Maternity Department of the Hospital were suggested. Very little discussion followed, however.

The time being limited, the meeting adjourned.

ST. JOSEPH HOSPITAL (Albuquerque.)

(Staff Meeting, Nov. 18, 1930.)

Dr. E. W. Johns presented the following case reports:

The case we have to present tonight is rather unusual. We first saw the patient October 18, at which time she had an exceedingly intense jaundice.

The past history was unimportant, except for a pelvic fracture one year ago in an auto accident. She had always been an unusually healthy and robust girl. Mother and father and one sister were living and well, as well as husband, to whom she had been married two years without pregnancy. Menses normal, and the last one occurred about a week before we saw her. She had no particular stomach trouble, as patient stated that, if she ate properly, she had no trouble, but did have occasional attacks of indigestion, which were attributed to indiscretions in diet. These had been a little more frequent since the auto accident, but none of a serious nature.

The present illness had begun one month ago with a severe nasal cold, since when she had not felt very well, but had been well enough to play golf two or three times a week, but would come home all tired out and felt tired most of the time. She had had a little more than usual indigestion during this time, but no attacks which gave much discomfort and no acute attacks of pain. She had noticed clay-colored stools for the past two weeks. About one week before we saw her, she menstruated, and it was more profuse than usual and she thought she might be miscarrying. Any attempt at abortion in any manner was emphatically denied. While menstruating, and five days before we saw her, she developed edema of the face and petechial hemorrhages of the skin, which were especially abundant on the arms and face. She quickly became very ill, with marked nausea and vomiting, together with severe prostration. The following day, severe jaundice appeared. When we saw her, four days later, there was no indication of the skin hemorrhages, except for a few scabs on the face, which were, perhaps, the result of scratching. The outstanding thing about her was her intense jaundice, which was intense everywhere, including the sclera and mucosa. She said that she had no pain and examination revealed only a very little tenderness on rather deep palpation in the gallbladder region and in the left upper quadrant in the region of the spleen. This latter pain was very slight, however; which is significant in the light of postmortem findings which will be given later. The abdomen had considerable distention from gas. There was almost a complete loss of liver dullness at first, which later partially returned. The heart was about 100 in rate. Blood pressure 120/70. There were no murmurs. P.M.I. was inside the mid-clavicular line. The lungs were clear. Respiration, 20. Reflexes normal. She was a very obese young woman about 20 years of age. Appeared to be fairly comfortable with moderate degree of prostration. Quite uncommunicative. Her unfailing response to the question as to how she felt, was always "fine." Leading questions always were answered as shortly as possible.

Temperature on admission was 98 degrees and at 8 p. m. that night a temperature of 100 degrees per rectum was recorded. After that the temperature ranged from 96.2 to 98.4 until the last 24 hours of life, when it went to 100.6 per rectum. Pulse range was very little, being between 90 and 100 very constantly until the last 24 hours, when it reached 130. Respirations were very constant, at 18 to 24. Van den Bergh's test was done once shortly after admission and found to be positive in both the direct and indirect reactions, and the Fouchet test was found postiive for hyper-bilirubinemia. The blood count was surprising, the first white count being 46,400, with 91 per cent polymorphonuclears, 7 per cent small lympocytes and 2 per cent transitionals. It was checked the following day and found to be 46,000. On the 25th, the white count dropped to 33,100; and on the 28th, to 18,600; while on the 30th, which was the day preceding her death, it had gone back again to 32,700. The nonprotein nitrogen on Oct. 26 was 176 mg. per 100 c.c. and the urea nitrogen 104 per 100 c.c., showing great retention of nitrogen. The urine, on several examinations, two of which were catheterized specimens, always showed a little albumin and many white blood cells, but no casts.

We instituted Lyon Meltzer gallbladder drainage and, after introducing about 200 c.c. of 30 per cent magnesium sulphate, finally got good drainage of bile. From this time on, her jaundice rapidly cleared and in a very few days her skin was a good color except for a somewhat ashen hue. The stools changed at once from a gray to a greenish yellow and remained so. She seemed to improve somewhat for a time and was able to retain a little light food and liquids but became quite anemic. The blood on the 28th showed 1,840,000 red blood cells and 36 per cent hemoglobin by Newcomer

method. On this date a transfusion was done and 600 c.c. of typed blood was given her. Type IV (Moss) blood was used, as she was Type III and such a donor was unavailable. She showed a good reaction but intractable nosebleed soon followed, which was not controlled until posterior packing was put into each nostril, in spite of heavy dosage of fibrogen and calcium chloride. The bleeding time on the 28th was 3 minutes and the coagulation time 4 minutes on admission and 5½ on the 30th, which was the day preceding death. Our diagnosis at first was obstructive jaundice, which, in the face of complete relief by Lyon Meltzer drainage, seemed conclusive except for the high white count. Acute yellow atrophy was considered strongly, because of the diminished liver dullness and the diphasic van den Bergh, and this was the diagnosis of a physician seeing her before we did. With the successful Lyon Meltzer drainage, this diagnosis became untenable. With the continued prostration, vomiting and high white count after the clearing up of the jaundice, we sought sources of toxemia. Her condition, however, was such that we did not think that an exploratory operation was warranted, in view of the lack of definite indications for such. In spite of the transfusion, together with rectal feeding and hypodermoclysis, she grew gradually weaker and died at 5 a. m. the morning of the 31st.

Consent for a postmortem examination was obtained and was performed by Dr. Warden. His findings were as follows:

The body is that of an adult, white female, apparently about 25 years of age, and about 5 ft. 3 in. tall, and rather obese. The face appears to be somewhat swollen. Red, frothy fluid exudes from the mouth. Both nostrils are occluded by blood-stained gauze packs. There is a small, surgical incision in the right cubital fossa which has been closed by adhesive tape. Needle punctures through the skin are seen at the lower margin of each breast and on the inner aspects of the thighs. The mucous membranes are pale. Some body heat is still present. Rigor mortis has appeared.

Considerable fat is encountered when a midline incision is made over the thorax and abdomen. The peritoneum is found to contain a normal amount of fluid and its surface in general is smooth and glistening. The appendix is slightly reddened and its peritoneal coat has lost some of its luster. The right fallopian tube is reddened and, when it is picked up, pus exudes from its fimbriated end. Stained smears of this pus reveal numerous pus cells, gram-negative rods, and gram-positive cocci in pairs and in short chains. A few flecks of fibrin are seen on the surface of the peritoneum adjacent to the fimbriated end of this tube. The gallbladder is distended and pressure upon the gallbladder fails to empty it. Several enlarged lymph nodes, the largest having a diameter of about 1.5 sm., are found along the course of the common bile duct.

When the thorax is opened about 750 c.c. of thin, red fluid is found in the left chest cavity. Within this fluid a few fibrin flakes are found. The pleural surfaces appear to be smooth and glistening. The lower lobe of the left lung is almost completely collapsed. There is only a small amount of fluid in the right chest cavity. Its surfaces are smooth and glistening. The pericardial sac contains a normal amount of fluid. Its surfaces are smooth and glistening. When the esophagus is opened its mucosa is seen to have a normal appearance. The trachea contains red, frothy fluid. Its inner surface is reddened. No abnormality is found in the aorta or in the inferior vena cava. The heart has a normal size. Its pericardial surface is smooth and glistening. Its chambers contain a small amount of clotted blood. Its valves have a normal appearance.

The right lung feels somewhat boggy, especially in its dependent portion, but crepitates throughout. Its surfaces are smooth and glistening. On surfaces made by cutting, a moderate amount of pink, frothy fluid exudes. The lower lobe of the left lung is small and does not crepitate. It has a fleshy consistency. The upper lobe crepitates throughout but feels somewhat boggy. The surface of the lower lobe is red. The pleura, however, appears to glisten. The surface of the upper lobe is gray. On surfaces of the lower lobe made by cutting, very little fluid exudes; surfaces of the upper lobe exude considerable pink, frothy fluid.

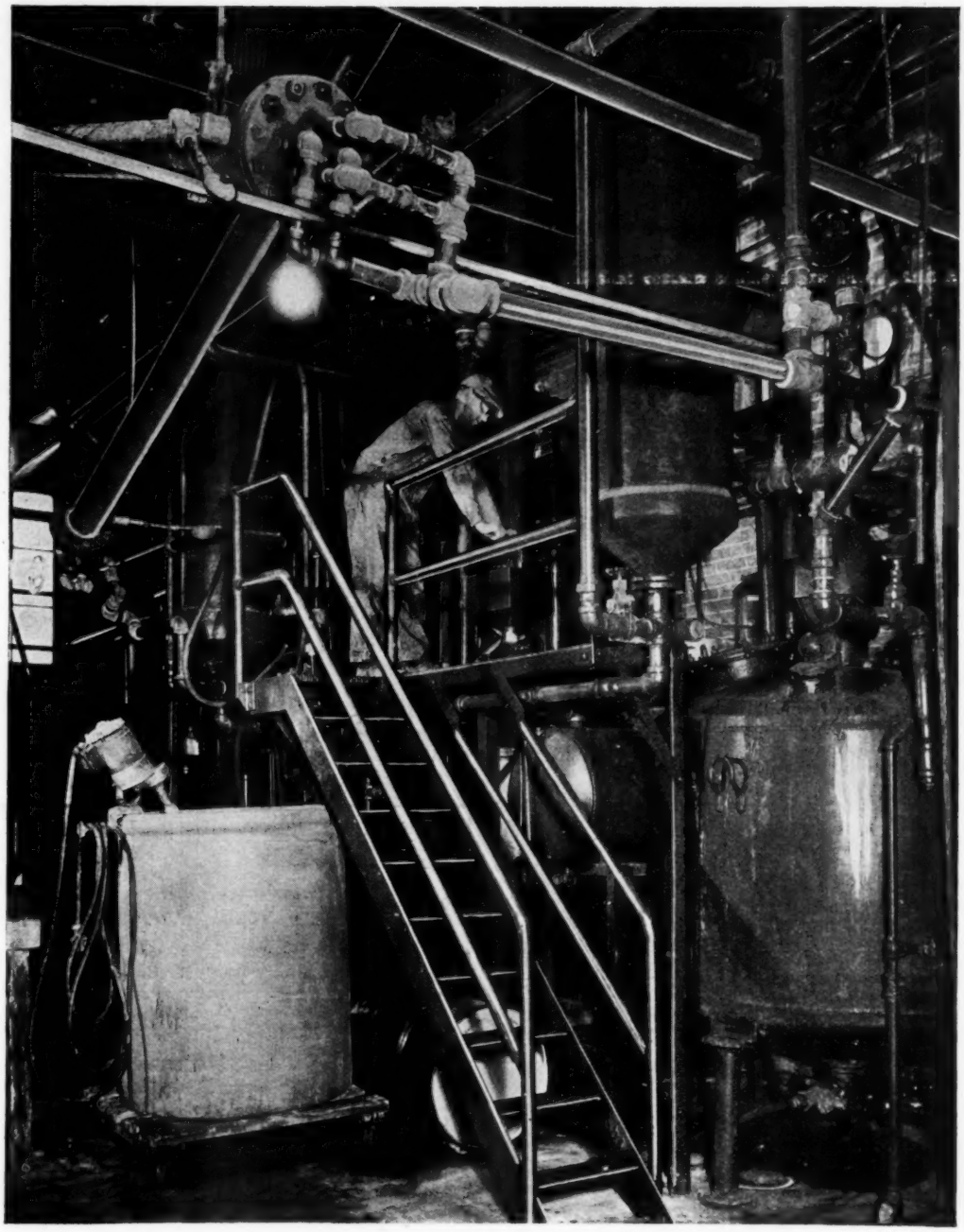
The kidneys have a normal size. The consistency of each is soft. The capsule of each strips readily, leaving a smooth surface. That of the left kidney is mottled, due to the presence of numerous, minute petechial hemorrhages. The renal markings are fairly well preserved in each kidney. Minute red points are seen, however, on the surface of each within the renal markings. The edges of cut surfaces do not roll out. In adrenals, ureters and bladder, no abnormality is found. The surfaces of the renal pelves, likewise, are free from gross pathology.

In the uterus and both ovaries, no abnormality is found. The left tube is grossly normal. The surface of the right tube is moderately reddened. When the tube is compressed, pus exudes from its fimbriated end.

The liver has a normal size. Its capsule is smooth and glistening. Its consistency is normal. On surfaces made by cutting, the centers of the lobules are seen to be stained green by bile, while the peripheries have a gray appearance. The gallbladder, when opened, is found to contain thick bile. No stones are found within it. No obstruction was demonstrated in the common bile duct. The large lymph nodes, however, previously described, lie in close approximation to the common bile duct.

When the spleen is displaced downward from the left diaphragm, a small sub-diaphragmatic abscess is encountered between the spleen and diaphragm. The surface of the spleen forms part of the wall of the abscess, and this portion of the surface is covered by a thick layer, apparently fibrin. Elsewhere on the surface of the spleen, several small miliary abscesses are encountered, which, when incised, are found to contain thick white pus. The spleen has about a normal size; its consistency is soft. The gastric surface of the spleen is adherent to the posterior wall of the stomach at the fundus.

The stomach, when opened, is found to contain about a liter of thin brown fluid. When this is washed out, and its mucosa inspected, a perforated ulcer is found, high up in its fundus and on the posterior wall adjacent to the spleen. It is this portion of the wall of the stomach which is adherent to the spleen. When the fibrinous adhesions which unite the two are broken, a small abscess containing thick, white pus is found. The ulcer opens into this abscess pocket. The ulcer itself has a diameter of only about 2 mm. and a normal mucosa extends up to the edge of the ulcer. Palpation of the wall of the stomach, bordering on the ulcer, reveals no thickening. On the outer surface of the stomach the peritoneum has disappeared over the portion of the stomach wall which forms



Stills and condensers employed in the production of Amytal and Sodium Amytal
View in the chemical department of Eli Lilly and Company, Indianapolis

ILETIN (INSULIN, LILLY), LIVER EXTRACT No. 343
PARA-THOR-MONE, MERTHIOLATE, EPHEDRINE PREPARATIONS

part of the wall of the abscess, and the muscle coat is exposed to view. In the remainder of the stomach no abnormality is found. The duodenal is normal. The remainder of the small bowel and the large bowel, while not opened, reveal no evidence of pathology when examined externally.

In the pancreas no abnormality is found.

In a culture made from the heart's blood, obtained under sterile conditions, no growth is obtained.

In a culture of pus from the right fallopian tube, there are gram-negative rods, gram-positive cocci, separately and in short chains, and long threads showing no branching, resembling the threads of some of the higher forms of bacteria.

Microscopic Examination

In stained microscopic sections of the liver, accumulations of greenish-brown pigment are seen in the areas surrounding the branches of the hepatic vein. In the portal areas, many of the cells contain large vacuoles and have a signet ring appearence. They have evidently been infiltrated by fat. A few similar cells are seen in the hepatic areas.

In sections of the kidneys, both are found to have a similar appearance. The majority of the glomeruli have a normal appearance. In a considerable number of the glomeruli, however, dilated capillaries are seen. Within the spaces surrounding some of the glomeruli, red blood cells are seen. Many of the tubules contain dense masses of red blood cells. No further abnormality is seen.

In sections of the lower lobe of the lung, the majority of the alveolar spaces are seen to be collapsed. The capillaries in the alveolar walls are distended by blood. Such alveolar spaces as are open contain pink-staining granular material.

In sections from the spleen taken from the region of the abscess, the capsule is seen to be thickened and to be infiltrated by both lymphoid cells and pus cells. Within the spleen iself the capillaries are dilated. At one margin there is an area in which cell outlines are seen but in which few nuclei are found.

In sections of a lymph node taken from the vicinity of the common bile duct, the lymph spaces appear to be dilated. There appears to be an increase in the number of endothelial cells.

Anatomic Diagnosis

Perforation of an ulcer in the fundus of the stomach, leading to a walled-off abscess between the stomach and the spleen, and to a small subphrenic abscess between the spleen and the left diaphragm. Secondary miliary abscesses in the spleen, and secondary acute hyperplastic lymphadenitis in the lymph nodes surrounding the common bile duct. Right acute suppurative salpingitis, with peritonitis localized to the region of the ostium of the right tube, secondary to the infection in the upper abdomen. Marked anemia, with probable generalized hemorrhagic diathesis. Recent hemorrhage from the nose. Multiple small hemorrhages into the tubules of both kidneys. Fatty in-

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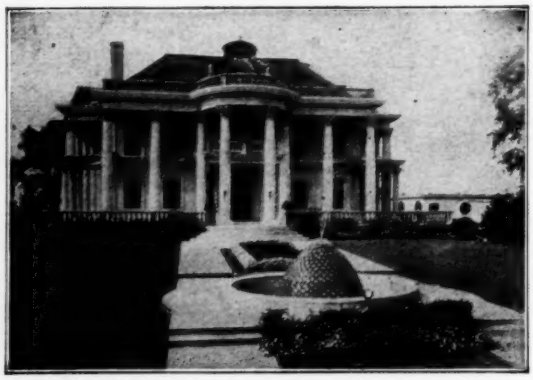

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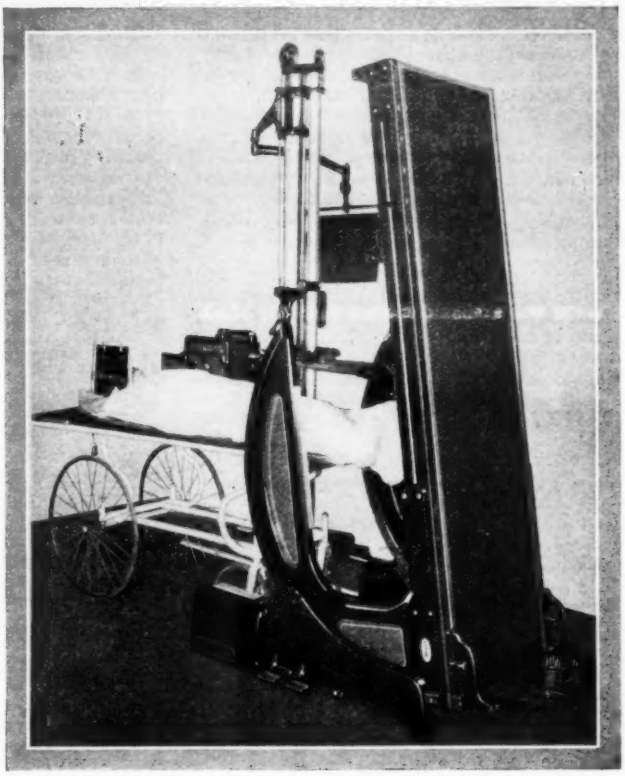

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filtration in a liver which is bile-stained from a recent jaundice (the jaundice probably being the result of compression of the common bile duct by an enlargement of the lymph nodes surrounding it.) Left hydrothorax, with compression atelectasis of the lower lobe of the left lung. Moderate edema of both lungs. Acute catarrhal tracheitis. Small surgical incision in the right cubital fossa (at the site of a recent blood transfusion. Multiple needle puncture marks in the skin (the result of subcutaneous administration of fluids.)

DISCUSSION

The finding of the perforated gastric ulcer was somewhat of a surprise to us, as we had nothing in either the history or the physical findings pointing toward it, except for the vomiting and the very slight tenderness in the splenic area. There was the diminished liver dullness, but, in view of the jaundice, it was associated with this condition. The lack of peritoneal symptoms was due to the very slow perforation into the culdesac between the stomach and spleen, which allowed adhesions to form before actual perforation occurred.

We have another case to report which is worth reporting because of its much more usual onset and the fact that it, too, was a perforated gastric ulcer. This case gave a history of severe stomach trouble for one year relieved by food taking. The present attack began shortly after a full meal at noon and was very sudden, with agonizing pain which was not relieved by morphine. After some delay the patient was brought 200 miles by auto to St. Joseph Hospital and was operated upon about 9 hours after the perforation occurred. A perforated ulcer in the anterior gastric wall, just proximal to the pylorus, was found. There were no adhesions. The convalescence was unusually smooth and uneventful.

These two cases are unusual, in that they are both gastric ulcers and were in the hospital at the same time, in view of the fact that all statistics show ulcers to be about 75 per cent duodenal and not over 25 per cent gastric, although of perforated cases about two-thirds are usually found to be gastric and, according to Bager, if duodenal and gastric ulcers in pyloric region are considered together, they will form about two-thirds of all perforations. They well illustrate the difference between a quickly penetrating ulcer without walling off adhesions, which represents about three-fifths of all ulcers, and the more slowly forming perforations which form adhesions. In the two-fifths of all ulcers where adhesions form, they are with the pancreas in about one-half of the cases, or one-fifth of all perforated ulcers; next most commonly the adhesions are with the left lobe of the liver, and much more rarely with other contiguous parts, as lymphatic glands, diaphragm, spleen, kidney, suprarenal capsule, omentum, colon, small intestine, gallbladder, sternum, or anterior abdominal wall. Our second case illustrates the more usual location of the peptic ulcer on the stomach wall, as Balfour states that 90 per cent are on or near the lesser curvature. Welch reports the location of 795 ulcers, in 566 cases, as follows, using a more definite localization than Balfour:

Lesser curvature	36.3%	
Posterior wall	29.6%	77.9%
Pylorus	12.0%	
Anterior wall	8.7%	
Cardia	6.3%	22.1%
Fundus	3.7%	
Greater curvature	3.4%	

Our first case falls in the fundus group of 3.7% of all cases, which is next to the lowest group, while our second probably would be classed by Welch with his pylorus group of 12 per cent.

There is considerable variation in the percentages which perforate in the different locations, which have been reported by Brinton as follows:

Posterior surface	2%
Pyloric region	10%
Middle portion of stomach	13%
Lesser curvature	18%
Cardiac extremity	40%
Anterior surface	85%

Our first case falls into the 2 per cent posterior surface of the stomach group, while our second probably would be grouped with the 10 per cent pyloric group, but, being on the anterior surface, it is at least akin to the anterior surface group with its 85 per cent of perforations.

These two cases are instructive in that they illustrate the two extremes of gastric ulcer perforation. The one, insidious and slow in onset, without serious symptoms; in fact, so innocuous that the patient did not consult a physician until 17 days before her death, and the symptoms were so disguised that perforated ulcer was not given more than passing consideration. The second, with very severe characteristic symptoms, making a probable diagnosis over long-distance telephone possible. The outcome of the two is very striking in that the one with the mild onset, apparently without serious illness, resulted in a fatal outcome to the patient, while the one with the

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sudden cataclysmic onset, with most apparent desperate illness of the patient, had a happy outcome.

Following a general discussion of the cases reported by Dr. Johns, Dr. L. S. Peters briefly outlined the history, the clinical course, and the post-mortem findings in a young boy recently seen by him, in which death was due to hemorrhage from a mediastinal abscess which had perforated through the trachea.

A series of motion pictures was then presented, illustrating the surgical treatment of peptic ulcer.

BOARD OF MEDICAL EXAMINERS OF ARIZONA

The following physicians have been granted license to practice medicine in Arizona, at the January, 1931 meeting of the Board of Medical Examiners:

DR. G. ARNOLD STEVENS, to be located at Clifton, Ariz. Graduate of the College of Medical Evangelists, Loma Linda, Calif., by reciprocity with the National Board of Medical Examiners.

DR. ALEXANDER McKEAN, Miami, Ariz., to be associated with the Health Department of Gila County. Graduate of Northwestern University, previously located at Salt Lake City. Reciprocity with Iowa.

DR. G. BYRON STEWARD, Stockton, Mo., graduate of St. Louis University (1920), licensed by reciprocity with Missouri.

DR. HOMER R. LATHROP, Casper Wyo., graduate of Rush Medical College (1901), licensed by reciprocity with Illinois.

DR. PAUL H. GUTTMAN, pathologist associated with the Grunow Memorial Clinic, Phoenix. Graduate of Washington University, St. Louis (1927), licensed by reciprocity with the National Board of Medical Examiners.

DR. JAMES OSCAR HICKS, Victoria, Texas, graduate of Kentucky School of Medicine (1926), licensed by reciprocity with West Virginia.

DR. ARTHUR H. WALL, Kansas City, Mo., graduate of Kansas City Medical College (1895), licensed by reciprocity with Missouri.

DR. GEORGE H. SPIVEY, Phoenix, Ariz., in charge of the work sponsored by the Rockefeller Foundation for the Maricopa County Board of Health. Graduate of University of Louisville Medical Dept. (1911), licensed by reciprocity with Mississippi.

DR. DAVID M. DAVIS, who will practice urology with the Grunow Clinic organization, Phoenix. Graduate of Johns Hopkins University (1911), licensed by reciprocity with Michigan.

DR. HENRY D. WATSON, Binghampton, N. Y. Graduate of University of New York (1896), licensed by reciprocity with New York.

DR. CHESTER L. WILSON, Yuma, Ariz. Graduate of University of Louisville Medical School (1929), licensed by examination.

DR. HAROLD L. SNOW, San Pedro, Calif. Graduate of Northwestern University, Chicago (1929), licensed by examination.

DR. B. P. STORTS, Jr., Ajo. Ariz. Graduate of University of Louisville (1929), licensed by examination. To be associated with the New Cornelia Copper Company.

DR. GEORGE F. C. FASTING, Tucson, Ariz., graduate of Rush Medical College (1926), licensed by examination.

DR. DOROTHY M. FRANKLIN, Tucson, Ariz., graduate of University of Illinois College of Medicine (1928), licensed by examination.

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The DALLAS SOUTHERN CLINICAL SOCIETY

Will hold its Third Spring Clinical Conference in Dallas, March 30th to April 3rd, 1931.

FEATURES:

17 DISTINGUISHED SPEAKERS including—

Fishbein	Hayden
Hirschman	Dean Lewis
Alvarez	Means
Berens	Chevalier Jackson
Caulk	W. A. Pusey
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DR. LOUIS B. BALDWIN, Phoenix, Ariz., to practice internal medicine with the Grunow Clinic. Graduate of College of Physicians and Surgeons of New York (1919). Licensed by reciprocity with New York.

DR. FRED P. PATTON, Tucson, Ariz. Graduate of Rush Medical College (1901), licensed by reciprocity with Illinois.

PERSONAL NEWS ITEMS

DR. H. D. ATWOOD, formerly located at Ajo, Ariz, on the staff of the New Cornelia Copper Company, has entered into the general practice of medicine in Phoenix, occupying the offices in the Heard Building formerly occupied by Dr. Couch. Dr. Atwood has taken charge of the work vacated by Dr. Calvert in Scottsdale, and is maintaining office hours in this suburban town, as well as in Phoenix.

DR. WM. C. TODT, of Kingman, Ariz., has been spending some weeks in Phoenix recuperating from a respiratory infection, which the rigors of the northern town would not allow to recover.

DR. R. O. LIEUALLEN, formerly Superintendent of the State Hospital in Phoenix, whose resignation from that position followed as the usual result of change in state administration, has accepted a responsible position with the California State Hospital at Norwalk, Calif.

DR. H. B. STEWARD, of Coolidge, Ariz., has been appointed physician for the State Prison at Florence, and has moved to the latter town to assume charge of his new duties.

DR. GEORGE W. STEPHENS, who was superintendent of the Arizona State Hospital under the last previous administration of Governor Hunt, has been re-appointed to that position and has taken charge of his old position at that institution.

DR. J. D. MAULDIN, of Phoenix, has been appointed Health Officer for Maricopa County, succeeding Dr. C. W. Brown, who gave up the office as the inevitable result of a new line-up of political forces the first of the year.

DR. JOHN R. EARP, formerly of Boulder, Colo., has been appointed Director of the Bureau of Public Welfare of New Mexico, succeeding Dr. Luckett. The reasons for Dr. Luckett's resignation have not been learned as yet, but the whole medical profession of the southwest will regret to lose him; the constructive work done by him in New Mexico during the ten years or more of service there has been outstanding. Dr. Earp comes to the office with many years of experience in executive work, having served among other responsible positions, as editor of Colorado Medicine.

DR. E. C. WILLS, of Phoenix, has opened offices at 704 N. Central Ave., where he will give special attention to electrotherapy. He has equipped his offices for medical and surgical diathermy, sinusoidal and galvanic electrotherapy, phototherapy by infrared or ultraviolet, electric vibration, massage, nerve regeneration and muscle training.

DR. JOHN WIX THOMAS has moved his offices from the Heard Building to his residence at 1217 N. Central Ave. He will observe his usual office hours at the new location, having remodelled a portion of the residence into office quarters.

DR. T. J. BOULDIN, of St. Johns, Apache County, is spending two months in Phoenix, as the state senator from that county. He was elected without opposition in the last general election.

DR. O. C. PATTON, of Ajo, is the member of the state legislature for the Ajo district of Pima County, and is spending the period of the legislature in Phoenix.

FIFTEENTH ANNUAL CLINICAL SESSION Of The AMERICAN COLLEGE OF PHYSICIANS

The Fifteenth Annual Clinical Session of the American College of Physicians will convene in Baltimore, Maryland, March 23-27, and in Washington, D. C., March 28. 1931. Those who attend the meeting will find ample in the way of clinical, laboratory, research and historical interest, to repay them for the time spent in making the journey. Dr. Sydney R. Miller, of Baltimore. President of the American College of Physicians, has prepared the program for the General Scientific Sessions, while Dr. Maurice C. Pincoffs, General Chairman, also of Baltimore, has arranged the program of clinics, demonstrations, entertainment. etc.

As an added feature of the Clinical Session this year, an additional day, March 28. will be spent in Washington, D. C., where a special program of clinics and inspection tours has been arranged under the auspices of the Medical Departments of the U. S. Army, U. S. Navy, U. S. Public Health Service and Georgetown University. Dr. William Gerry Morgan is acting as Chairman of the Washington Committee.

The entire program of the Clincal Session is characterized by new subjects, new authors and wide geographic representation. It is significant that the Committees have attempted carefully to avoid repetition of subjects and authors, as has so often been the case in previous years. On the General Scientific Programs there will be forty-five or fifty selected formal papers. Symposia on blood diseases, oxygen therapy, diseases of the liver, recent advances in endocrinology with particular reference to the newer work on supra-renal extracts, myocarditis, and several other subjects have been arranged. At Baltimore's many modern and excellently operated hospitals, clinics, ward-walks, laboratory demonstrations and the like will be held. Johns Hopkins Hospital and Medical School, under Dr. Alan M. Chesney, Dean, and a specially appointed Committee, will place at the disposal of the College all of its facilities and offer a program of great interest. Additional hospitals, such as the Union Memorial Hospital, St. Agnes Hospital, at which Dr. Joseph C. Bloodgood does so much of his work, the Municipal Hospitals, and several of the more private institutions, such as the Howard A. Kelly Hospital, noted particularly for its radium activities, and the Sheppard and Enoch Pratt Hospital, which is one of the most modern dealing wiht psychiatric problems, and many others will provide programs of clinics.

Hotel headquarters will be at the Lord Baltimore Hotel, while general headquarters, at which the registration of members, commercial exhibits and all general sessions will be held, will be The Alcazar, Cathedral and Madison Streets, Baltimore. Transportation on the Certificate Plan of reduced fares will be available to all physicians and dependent members of their family from all parts of the United States and Canada. A special program of entertainment has been arranged for visiting ladies. The Convocation for the induction of new members, as Masters or Fellows, will be held on Wednesday evening, March 25, and the Annual Banquet will be held on Thursday evening, March 26. The Business Meeting, at which reports of administration and elections for the new year will take place, will be held during the forenoon of Thursday, March 26.

Mr. E. R. Loveland, 133-135 S. 36th Street, Philadelphia, is the Executive Secretary of the College, and it is to him that requests for further information or programs should be addressed.

DALLAS SOUTHERN CLINICAL CONFERENCE

The third Annual Spring Clinical Conference of the Dallas Southern Clinical Society will be held in Dallas during the week of March 30th. Invitations to attend are being sent to all orthodox physicians of the Southern States and Mexico.

The program for the week is built around the lectures and hospital demonstrations of seventeen guest lecturers, among whom are Dr. Walter C. Alvarez of Rochester, Minnesota, Dr. Louis J. Hirschman of Detroit, Drs. Morris Fishbein, Russell Wilder, C. G. Grulee, L. J. Pollock, A. A. Hayden and W. A. Pusey of Chicago, Drs. Chevalier Jackson and H. K. Pancoast of Philadelphia, Dr. Dean Lewis of Baltimore, Dr. Jeff Miller of New Orleans and others. Ninety-six hours of organized post-graduate work is being offered by local men in addition to the general assemblies and hospital clinics. The Baker Hotel is headquarters for registration and all assemblies.

THE DOCTOR IN COURT, by Edward Huntington Williams, M.D.; The Williams and Wilkins Company; Baltimore; 1929.

The physician may at any time be called to testify in court. The better the witness, because of his knowledge, the more likely will be the opposing attorney to attempt to embarrass him.

The methods by which a witness may be attacked in cross-examination are devious and numerous.

Dr. Williams gives actual court room experiences, and shows the reader the types of situations which may arise. No book can supplant court room experiences and make the novice the equal of the experienced witness; a careful study of this book, however, must make the physician better prepared to testify when compelled to do so.

The small price of the book will be returned perhaps many times to the physician who is familiar with its contents before appearing in court.

The author writes entertaingly. The publisher has done excellent work. There are relatively few typographical or grammatical errors. The book can be read in an evening.

Every physician should own this book and give it frequent re-readings. O. H.B.

Eye, Ear, Nose and Throat man in active practice and good standing, experienced and accomplished, desires location or association in New Mexico or Texas for the benefit of a warmer climate. Add. Box W, Southwestern Medicine.

The Dugdale Libel Suit.—Frederick Dugdale, M. D., of Lowell and Boston had his license to practice medicine in Massachusetts revoked April 18, 1929, for "gross misconduct in the practice of his profession." The Lowell (Mass.) Sun published an account of the hearing connected with the revocation proceedings and stated that the charges against Dugdale grew out of the alleged professional misconduct on his part in the treatment of a patient who subsequently died of cancer. As a result of this publication Dugdale brought suit against the publisher of the Sun, charging libel and demanding damages. The case came to trial and the jury brought in a verdict in favor of the newspaper. For some years Dugdale advertised as a "specialist" in "skin, blood and nervous diseases" and operated a mail order "rheumatism cure"; later he was one of the disciples of the Abrams cult. For some years Dugdale had his own "cancer cure." Still later, as the Koch "cancer cure" became a money-maker, Dugdale became a Koch disciple and in the libel suit the Koch "treatment" figured largely. (Jour. A.M. A., November 15, 1930, p. 1523.)

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OLD MOTHER HUTTON, the Shropshire herb-woman, guarded her secret jealously. Had not her herb tea relieved an Oxford dean of his dropsy, when all other remedies had failed? To satisfy his curiosity, Dr. William Withering sought out the old woman, nearly 150 years ago. At first she stubbornly refused to talk to him. But finally one argument prevailed. There was the clink of golden sovereigns in Mother Hutton's palm, and the cherished recipe was his!

Withering saw that the crudely written scrawl called for twelve common herbs and plants. After long and patient trial he found eleven of them medicinally worthless. But in the twelfth, the purple foxglove, he found a new and powerful drug, now more commonly known as digitalis.

Physicians welcomed Withering's contribution to medicine, but they soon found that the use of digitalis was attended by a serious difficulty. Extracts made from the drug were by no means uniform in potency. Some were too weak; others were much too strong for safety.

For over a century, physicians and pharmacists grappled with the problem of making uniform extracts of digitalis. Then, in 1898, exactly 113 years after Withering's discovery, the Parke-Davis medical research laboratories announced the first *standardized* tincture of digitalis.

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